A

PRACTICAL TREATISE

ON

THE DISEASES OF THE LUNGS, AND HEART,

INCLUDING

THE PRINCIPLES OF PHYSICAL DIAGNOSIS.

BY

WALTER HAYLE WALSHE, M.D.

PROFESSOR OF THE PRINCIPLES AND PRACTICE OF MEDICINE, AND OF CLINICAL MEDICINE IN
UNIVERSITY COLLEGE, LONDON; PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL,
CONSULTING PHYSICIAN TO THE HOSPITAL FOR CONSUMPTION.

> ". . . . Who knows but that one may discover the works performed in the several offices and shops of a man's body, by the sounds they make, and thereby discover what instrument or engine is out of order?"
>
> R. HOOK, 1705.

LONDON:
TAYLOR, WALTON, & MABERLY.
UPPER GOWER STREET, AND IVY LANE, PATERNOSTER ROW
1851.

616.24
WAL/P

LONDON:
BRADBURY AND EVANS, PRINTERS, WHITEFRIARS.

TO

JOHN FORBES, M.D., F.R.S.,

AND

NEIL ARNOTT, M.D., F.R.S.,

IN

ADMIRATION OF THEIR EMINENT PROFESSIONAL CHARACTER,

AND

IN GRATITUDE FOR FRIENDSHIP AS DISINTERESTED

AS UNCHANGING.

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PART I.

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PHYSICAL EXAMINATION OF THE LUNGS, HEART, AND GREAT VESSELS.

INTRODUCTION.

THE existence of disease involves that of anatomical change, not only in the part originally and chiefly affected, but also in the structures immediately adjoining. There are a few apparently pure dynamic diseases, forming doubtful exceptions to this proposition; but, admitting their reality, they are not of sufficiently great importance to affect the general truth.

The anatomical changes thus arising may or may not be capable of accurate discrimination during life. When they can be so discriminated, experience has shown that their detection is not so much accomplished by means of the vital functional derangements of the organs implicated, as by the aid of various alterations in the physical properties of those organs,—as, for example, their density, their faculty of generating and of conducting sound, &c. So invariably do these alterations bear a certain and fixed relation to the nature of the anatomical conditions with which they are associated, that the discovery of the former is conclusive as to the existence of the latter. And not only the nature, but the precise limits and the precise degree of these conditions are disclosed by the alterations referred to, which, for these reasons, constitute their *Physical*

Signs. Interpreted by the observer, and not by the patient,—incapable of being feigned or dissembled,—estimable in degree and extent often with mathematical precision,—susceptible of indefinite refinement,*—physical signs, like the whole class of objective phenomena of disease, are of materially greater clinical value than its subjective symptoms. Physical signs are, then, the true indices of the nature, extent, and degree of organic textural changes, and may be regarded as instruments of pursuing morbid anatomy on the living body. But just as their significance is sure and precise, so is the difficulty of mastering their theory and practice positive and great; and hence it is that Physical Diagnosis has gradually acquired for itself the importance of a special art.

The means by which the existence and nature of physical signs are discovered, are called *Physical Methods of Diagnosis;* and these methods vary with the properties, position, and functional relations of the organs examined. The diseases of the organs of respiration and of circulation are those of which the physical *signs* are best understood and most readily ascertained; the *methods* employed in their detection are:—

I. INSPECTION; II. APPLICATION OF THE HAND; III. MENSURATION; IV. PERCUSSION; V. AUSCULTATION; VI. SUCCUSSION.

These methods are, as nearly as is possible, applied to the organs themselves of which we desire to ascertain the condition,—to the external surface corresponding to them, when inapplicable to themselves. But the absence or presence of disease in the different thoracic organs, and, if it exist, its nature, may sometimes be indirectly inferred by employing these methods in

VII. THE DETERMINATION OF THE SITUATION OF SUR-

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* In the present state of knowledge it must be confessed that physical signs directly reveal only physical conditions; but it is very probable that in time they will reveal the nature of disease too. The fundamental sounds of lung, *in situ,* solidified by cancer, by tubercle, or by simple exudation-matter, for instance, cannot be the same; though by the percussor of the present day no special differences can be caught.

ROUNDING PARTS AND ORGANS,—which may consequently be considered an additional method of physical diagnosis.

All these methods agree in the general character of their direct and indirect objects. The *direct object*, the physician has in view with all, is the just appreciation of the sensations they furnish, and these are nothing more than the physical signs already spoken of; the *indirect object*, the reference of these signs to the material states upon which they depend.

The general description of each of these methods must comprehend an inquiry into:—Its nature; its direct or immediate object; the manner of practising it; the conditions which are discovered by its means in the healthy state; such deviations from the ordinary standard of these conditions as are, nevertheless, compatible with health; the deviations from that standard which are actually morbid and constitute signs of disease.

With the view of localising physical signs as precisely as possible, the surface of the chest has been divided into artificial regions; but as the assignment of limits to these regions is altogether arbitrary, it is not to be wondered that the boundaries adopted by different writers vary. Simplicity, as far as is compatible with the main object, should be especially aimed at in all such topographical arrangements; but it scarcely appears possible, without a sacrifice of utility, to make the number of divisions less than in the following plan. They are designed to correspond, as far as is attainable, with important internal regions or outlines.

The chest is divided into anterior, lateral, and posterior regions. The *anterior* are called: supra-clavicular, clavicular, infra-clavicular, mammary, infra-mammary, supra-sternal, upper sternal, lower sternal. The *lateral* regions are: the axillary, the infra-axillary. The *posterior* series comprises: the upper scapular, the lower scapular, the infra-scapular, and the inter-scapular. Of these regions the supra, upper and lower sternal are single; all the rest are double.

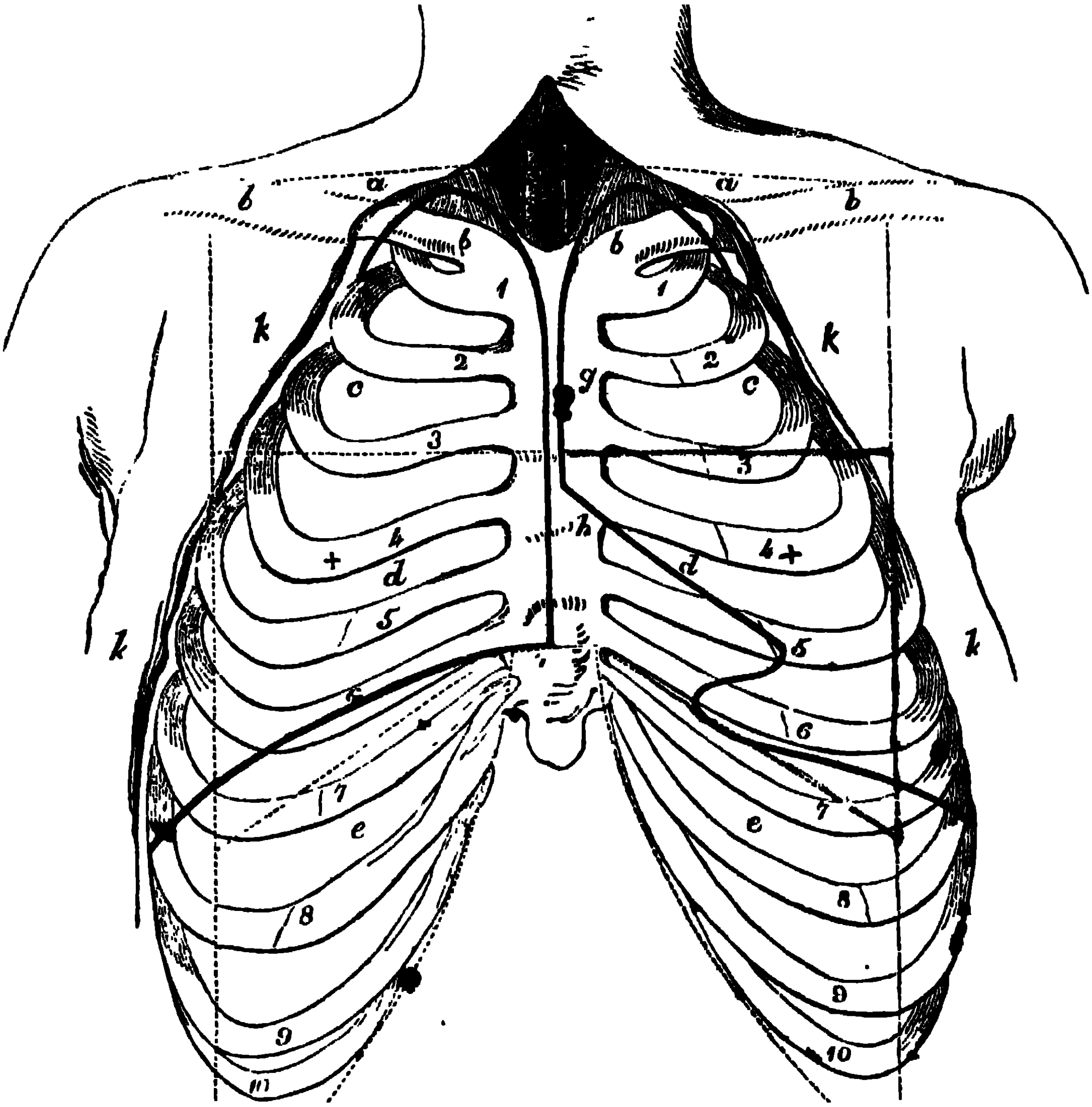

Diagram exhibiting the anterior regions of the chest, with their relationship to the ribs, and also the position of the edges of the lungs in calm (phrenic) respiration. 1 to 10 inclusive, ribs; *a*, supra-clavicular region; *b*, clavicular region; *c*, infra-clavicular; *d*, mammary; *e*, infra-mammary; *f*, supra-sternal; *g*, upper sternal; *h*, lower sternal; *i*, trachea; *k*, integuments The dotted lines indicate the boundaries of the various regions; the thick lines correspond to the outlines of the lungs, which rise to a maximum height of one inch and a quarter above the clavicle; (at the apex the *outer* border is carried to the line of *full* inspiration); + +, the nipples. Taken from an adult male, the trachea being tied after moderate insufflation of the lungs.

The boundaries of these regions, and the more important structures and portions of organs corresponding to them, either within the chest or on its confines, may be stated as follow:—

Supra-clavicular.—Above, a line drawn from the outer part of the clavicle to the upper rings of the trachea; below, the clavicle; inside, the edge of the trachea. Here are found the triangular apex of the lung, portions of the subclavian and carotid arteries, and of the subclavian and jugular veins; the first rib contributes to form a sort of floor for the region.

Clavicular.—Corresponds in its outline to the inner half of the clavicle. Beneath the bone lies on both sides lung-substance; on the right side (at the sternal articulation) the arteria innominata just reaches the inner confines of the region, while the subclavian artery crosses it at its outer end; on the left side the carotid and subclavian arteries lie deeply, almost at right angles with the bone.

Infra-Clavicular.—Above, the clavicle; below, the lower border of the third rib; outside, a line falling vertically from the acromial angle (formed by the clavicle and the head of the humerus); inside, the edge of the sternum. Within these limits are placed the upper lobe of the lung, on both sides; on the right side, in addition, the superior cava, and a portion of the arch of the aorta; on the left, the edge of the pulmonary artery. The vessels on both sides lie behind the costal cartilages of this region; and the second right cartilage may be familiarly termed the "aortic" cartilage, the second left the "pulmonary." On the left side, the inferior border of this region corresponds to the base of the heart. The bifurcation of the trachea taking place behind the arch of the aorta, on the level of the second rib, the main bronchus on either side is found in this region, the right behind, the left a little below, the second costal cartilage.

Mammary.—Above, the lower border of the third rib; below, the sixth rib; outside, a vertical line continuous with the outer border of the infra-clavicular region; inside, the edge of the sternum. The contents of this region differ materially on the two sides. On the right side, the lung lies throughout immediately under the surface, extending downwards to the sixth rib, where (its inferior border lying almost horizontally) it nearly corresponds to the lower edge of the region. The right wing of the diaphragm and the liver commonly rise to the fourth interspace. The line of separation of the upper and middle lobes of the right lung passes obliquely upwards and backwards from about the fourth cartilage; that of the middle and lower, in this direction from the fifth interspace. A portion of the right auricle and the upper and right angle of the right ventricle lie between the third and fifth ribs, close to the sternum. On the left side, the edge of the lung passes obliquely downwards and outwards from (usually) the fourth cartilage, leaving a free space of variable size for the heart,—thus reaches the fifth rib, then curves inwards and downwards to opposite the sixth rib or interspace (within the vertical line of the nipple), whence it passes nearly horizontally outwards. The anterior point of division of the lobes of this lung lies about the fifth interspace, below the nipple. The left auricle and left ventricle, with a small portion of the right ventricle about the apex, lie within this region.

Infra-Mammary.—Above, a line slanting outwards from the sixth cartilage; below, a curved line corresponding to the edges of the false ribs; outside, the outer edge of the mammary region prolonged; inside, the sternum at its inferior angle. On the right side the liver (with the lung protruding in front on full inspiration) occupies this region. On the left side lie the stomach and anterior edge of the spleen, which rises as high as the sixth rib; in the inner part of the region there is generally a portion of the left lobe of the liver, lying in front of the stomach.

Supra-Sternal.—A small region, more or less hollow, bounded below by the notch of the sternum, and laterally by the sterno-mastoid muscles. The trachea fills it almost completely; it contains no lung; the innominate artery lies at its lower right angle; and in some persons the arch of the aorta reaches its lower border.

Upper-Sternal.—Corresponds to that portion of the sternum lying above the lower border of the third rib. Here are found the left (and a small portion of the right) innominate vein; the ascending and transverse portions of the arch of the aorta; the pulmonary artery, from its origin to its bifurcation; the aortic valves, near the lower border of the third left cartilage,—the pulmonary a little higher than these, and quite at the left edge of the bone; and the trachea, with its bifurcation on the level of the second ribs. The inner edges of the lungs, coming slantingly downwards, usually almost join on the middle line opposite the same ribs, and continue thus united for the rest of the region, lying immediately under the sternum.

Lower-Sternal.—Corresponding to the remainder of the sternum, this region contains the main part of the right ventricle, and a small part of the left; the line of union of the heart and liver (with the diaphragm intervening); the edge of the right lung descending vertically along the middle line, and, at its upper part, a small portion of the left lung; and inferiorly, and deeply-seated, a portion of the liver, and sometimes of the stomach. The tricuspid and mitral valves (the latter somewhat posterior) lie at mid-sternum opposite the upper edge of the region, or a little below this.

Axillary.—Extending from the point of the axilla above, to a line continuous with the lower border of the mammary region below, and in front from the posterior border of the infra-clavicular and mammary regions to the external edge of the scapula behind, this region corresponds to the upper lobes of the lungs, with, deeply-seated, the main bronchi.

Infra-axillary.—Bounded above by the axillary region, anteriorly by the infra-mammary, posteriorly by the infra-scapular, and below by the edges of the false ribs, this region contains on both sides the lower edge of the lung sloping downwards from before to behind, with, on the right side, the liver, and, on the left, the spleen and stomach.

Upper scapular and lower scapular.—Have the same boundaries as the fossæ of the scapula, and correspond to lung-substance.

Infra-scapular.—Above, the inferior angle of the scapula and the seventh dorsal vertebra; below, the twelfth rib; outside, the posterior edge of the infra-axillary region; inside, the spine.

Immediately underneath the surface, as far as the eleventh rib, lie the lungs; on the right side, the liver from the level of the rib just named, to the lower edge of the region; on the left, the intestines, occupying some of the inner part of the region, and the spleen of the outer. Close to the spine, on each side, a small portion of the kidney encroaches on this region (more on the left than the right side); and along its inner edge, on the former side, runs the descending aorta.

Intra-scapular.—Occupying the space lying between the inner edge of the scapula and the spines of the dorsal vertebræ from the second to the sixth, this region contains lung-substance on both sides, the bifurcation of the trachea and the main bronchi, the bronchial glands, with, on the left side, the œsophagus, and (from the fourth vertebra downwards) the descending aorta.

PREFACE.

In the first of the two Parts, into which this work is divided, an attempt is made to describe the facts and principles of Physical Diagnosis in their applications to the Lungs, Heart, and great vessels. Although in the main confining himself to a description of physical signs, the Author has occasionally stepped aside to consider their mechanism,—yet not without the full consciousness, that in the present state of thoracic acoustics (and, indeed, of acoustics in general), all efforts of the kind must be merely tentative. If, on the one hand, the theories of the immortal discoverer of auscultation do not, in all instances, bear the test of clinical scrutiny, those of his German critics seem neither to be always in harmony with the principles of acoustics, nor accordant with the results of actual experiment. Whether such modifications as the Author has ventured to suggest in the theory of certain auscultatory signs, be of more satisfactory character, the verdict of his professional brethren can alone decide.

In the second Part, the symptoms, physical signs, diagnosis, and treatment of the chief diseases of the Lungs, Heart, and Aorta, are clinically described. The Author's design being to furnish, within a moderate compass, a guide to the detection and treatment of those diseases, he has systematically avoided (unless where some particular object obliged him to deviate from his plan) all inquiries into their general or special pathology.

When morbid anatomy was first seriously cultivated, the effects on medical practice were deeply depressing. It was naturally felt that the anatomical cure of the textural changes the scalpel revealed, was an impossibility. But a reaction has fortunately taken place; the conviction has gradually been forced upon observers, that many diseases texturally incurable, are mitigable by treatment to such a degree in their local and constitutional ill effects, as to be rendered comparatively innocuous. And of no diseases is this more true than of the chronic pulmonary and cardiac classes. Singularly happy as, at the time he wrote, appeared the motto chosen by Corvisart for his celebrated work on Diseases of the Heart,—"hæret lateri *lethalis* arundo;" a phrase so hopeless is at the present hour infinitely less appropriate.

CHAPTER I.

PHYSICAL EXAMINATION OF THE LUNGS.

SECTION I.—INSPECTION.

BY inspection of the chest, as a method of physical diagnosis, is understood simply the ocular examination of its external surface; by inspection are ascertained the conditions of exterior *form* and *size* of the cavity, and of the *motions* of its walls. The *form* of the chest is to be considered in respect of its *general configuration*, and the *shape of its various parts*. The *size* of the cavity is less important considered as a whole, than as composed of two divisions; the relative dimensions of these being the point of real consequence. The *motions* of the chest are *general* and *partial*: the *general* motions being those of *expansion* and of *elevation*; the *partial*, consisting of a *movement of the ribs in respect of each other*.

In order to insure correct results from inspection of the chest, the following precautions are to be observed:—that the light be good; that the surface examined be fully exposed; that the patient's muscles be relaxed, and all physical restraint removed; and, above all, that the plane on which he lies, stands, or sits, be perfectly even. When the patient's state allows the observer the choice of the three postures just mentioned, the sitting ought decidedly to be selected. Inspection should be practised anteriorly, posteriorly, laterally, and from above downwards,—in the latter direction particularly, as a means of roughly ascertaining the antero-posterior diameter of the chest. Under all circumstances, it is of the last importance that the two sides, both generally and in their various corresponding parts, be closely *compared.* This observation applies with the same force to all other methods of physical examination; without *comparison* of corresponding regions the utility of this kind of investigation would be very materially diminished. But in order that such comparison shall not be fallacious, it is essential that the observer should be fully alive to the numerous physical differences which naturally exist in corresponding parts of the two sides.

A. *Form.*—I. *In Health.*—The form of the chest of persons who have never had any affection of the thorax itself or its contents may be *regular,* or more or less *irregular.*

A chest *regularly* formed presents *to the eye* (abstraction being made of every thing except its own immediate integuments) a cone having its narrow end uppermost; its transverse diameter obviously exceeding the antero-posterior; its two sides symmetrical, both generally and in their different parts; the supra-clavicular spaces very slightly depressed; the lower sternal region hollowed out in proportion to the stoutness of the subject; the infra-clavicular regions gently convex; the angles, formed by the union of the false ribs and middle line anteriorly, very nearly equal,—that on the right side being slightly the more obtuse; the intercostal spaces visible in inspiration and expiration, unless the individual be very fat; the lateral surfaces of the chest equally distant from the median plane,—as likewise the nipples, which are on the same level, that of the fourth rib or fourth intercostal space; the different regions of the chest, considered in themselves, regularly shaped; the shoulders on the same level; the spine either perfectly straight, or inclining very slightly to the right at mid-back; and the vertebral sulcus, moderately concave from above downwards, more or less deep according to the fatness or thinness of the individual.

But it is comparatively rare to find a chest having in all respects the characters now enumerated. Certain deviations of form, perfectly compatible with a healthy state, both of the thoracic organs, and of the body generally, are of extremely common occurrence. It would follow, indeed, from the investigations of M. Woillez,* that a *regularly* formed chest (as just described) exists in scarcely more than one of every five adult males taken indiscriminately. The irregularities or *heteromorphisms*,† which render the chest non-symmetrical, while they are perfectly compatible with health, are by this author termed *physiological;* the name *pathological* being applied to those that are the manifest results of disease.‡

It is obvious that the chief, almost the sole, clinical importance of these "physiological" departures from regular form consists in the chance of their being mistaken for alterations of shape dependent on disease. Their frequency indicates the necessity of acquaintance with them: in 197 cases there existed 251 such heteromorphisms; 144 of these occurring in 111 persons who had had thoracic disease, 107 in 86 individuals who had all their lives been perfectly free from such disease.

Physiological heteromorphisms may be congenital or acquired, and general or partial. The *general* are those in which the natural relations of the different diameters of the chest are altered; the *partial* consist of local defects of symmetry, exercising no influence on the general shape of the thorax.

Again, certain local irregularities of form may be either of physiological or of pathological origin: disease may produce in one chest precisely the same alteration of shape that accidental circumstances, in no wise impairing health, effect in another.

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* Recherches Prat. sur l'Inspection et la Mensuration de la Poitrine. Paris, 1838.

† From ἕτερος other, and μορφὴ form.

‡ *Regularly* formed chests are more common before than after the age of thirty, and in persons who follow sedentary pursuits or trades requiring little muscular exertion, or who have never laboured under thoracic disease, than among other individuals. The previous occurrence of such disease does not, of course, necessarily imply the existence of irregularity of form.

When a deviation of form, which may be thus either morbid or not, presents itself, its mode of origin can only be positively determined by the absence or presence of other signs denoting subjacent disease, or by the previous history of the individual showing that he has or has not suffered from pectoral complaints.

II. *In Disease.*—Alterations of form and of position of the whole thoracic surface, or of its parts, if considered in regard of *their physical characters*, may be referred to the following species: —(α) Expansion and Bulging; (β) Retraction and Depression; (γ) Procidentia and Elevation; (δ) Curvature; (ε) Distortion.

(α) *Expansion* signifies a change of shape of the chest, in which one or both of its sides is generally prominent; *bulging*, a local or circumscribed expansion, the remainder of the thoracic surface being either in the natural state or affected with some other species of irregularity. Expansion of one side, produced by some force acting from within outwards (the elasticity of the lung having been first destroyed), is best seen in cases of abundant pleuritic effusion, with or without pneumonia; in pneumo-thorax, hydro-pneumo-thorax, and general vesicular emphysema; less clearly in hypertrophy of the lung, intra-thoracic tumors, and extensive hæmo-thorax. Simple pneumonia and hydro-thorax have not yet been proved to produce it; nor are any affections of the heart or great vessels capable, even as matter of theory, of doing so. Expansion of either side is never a physiological heteromorphism.

Bulging occurs at either base in pleuritic effusion and in pleuro-pneumonia; in emphysema appears above and below the clavicles, and has been observed in those regions in cases of simple pneumonia of the apex; exhibits itself in various sites in cases of circumscribed pleurisy and intra-thoracic tumor; in the right infra-axillary region in cases of enlarged liver, and in the left of enlarged spleen; in the mammary and lower sternal regions in pericardial effusion and hypertrophy of the heart, and in the upper and central parts of the chest in cases of aortic aneurism. But, on the other hand, bulging frequently occurs as a natural condition in the following positions: the right back inferiorly; the left front inferiorly, with or without twisting forwards of the free edges of the ribs; the upper sternal region; the second costal cartilages, either or both; and the left sterno-mammary regions. Such bulgings simulate those produced by pericardial effusion, aneurism of the aorta, pleuritic effusion, &c.

(β) *Retraction* and *depression* are the converse states of expansion and bulging: the former, a general sinking of the walls on one side; the latter, a similar condition limited to one spot or region. *Retraction* never exists without reduction of size of the lung, produced either by extrinsic pressure or by changes in its own substance. Pressure is essentially concerned in cases of pleuritic effusion: the lung reduced to a small bulk by the pressure of accumulated fluid, deprived of its elasticity, and bound down by pseudo-membrane, is unable to resume its original volume on the removal of the fluid by absorption,—the side consequently yields inwards, under the weight of the atmosphere. The false membrane aids materially, through its characteristic force of contraction, in producing this result, by diminishing the bulk of the lung,—not, as might be supposed, by actually dragging the wall of the chest inwards. Changes of the lung-substance, reducing its bulk, occur in tuberculous disease, the absorptive period of pneumonia, in cancerous and certain exudative infiltrations of the lung, and in collapse and atrophy of the organ consequent on the inaction entailed by pressure on its main bronchus by enlarged glands, tumor, aneurism, &c. *Depression* attends the same morbid states, when more limited in extent and influence. In estimating the clinical value of depression, the observer must remember that it sometimes occurs physiologically in the lower sternal region, and (symmetrically) in the infra-mammary regions.

(γ) *Procidentia* is that state in which the position of a part is lower than natural; *elevation*, that in which it is higher. Examples of *procidentia* are seen in the lowered position of the shoulder, the ribs (especially externally) and the nipple in chronic pleurisy with retraction. The shoulders are not always naturally on the same level, however; and the left nipple is, in healthy persons, frequently lower than the right. I once met with *elevation* of the shoulder, on the *same* side as retraction of the parietes from chronic pleurisy.

(δ) *Curvature* signifies that deviation of the various axes of a part in which some degree of regularity of form is retained; *distortion*, a displacement of the same kind fundamentally, but one in which the deviations are so numerous and so considerable that all trace of regular shape is lost. The spine, sternum, clavicles, and ribs, are subject to the former of these displacements in connexion with disease of the subjacent organs. Thus the dorsal spine becomes laterally curved (the convexity to the sound side) in cases of chronic pleurisy with retraction; the ribs, in extreme cases of the same disease, undergo such torsion on their longitudinal axes, that their upper edges become external; the sternum yields sidewards under the pressure of tumours, and the clavicle twists downwards and inwards in some cases of tuberculous destruction of the apex. Some slight deviation of the dorsal spine to the right, I have found to be more common than perfect straightness in male adults with sound chests; and the sternum sometimes naturally inclines to one side.

B. *Size*.—No positive rule can be laid down respecting the natural visible dimensions of the thorax in proportion to that of the body generally: the proportion varies widely in different individuals enjoying robust health.

There is no *visible* inequality of size in the two sides of a well-formed thorax. Numerous deviations from the natural relative dimensions of the different parts of the thorax occur in consequence of disease; but as they are always to be more accurately estimated by measurement than by inspection, and in some cases only to be ascertained at all by means of the former, the consideration of their different varieties is deferred to the section on MENSURATION.

C. MOTIONS, IN HEALTH.—(*a*) The *general* motions, or those in which the entire thorax is concerned, are of *expansion* and of *elevation*. In health these two kinds of motion are so intimately associated and agree so closely in proportional amount, that it is unnecessary to consider them separately; in certain states of disease they are very differently affected.

During *inspiration* the walls of the chest diverge from their central axis,—the anterior ribs passing somewhat forwards, the lateral outwards, and the posterior backwards, from that axis (*expansion-motion*). At the same time the anterior walls rise upwards (*elevation-motion*). Both these classes of motion are, in the majority of people, slightly more active at the middle than the commencement or close of inspiration, but free from all jerking inequality of rhythm. They are freer and more extensive at the lower parts of the thorax in males, at the upper in females;* and are not perfectly equal in amount on both sides of the chest at all levels. In ordinary breathing these motions are in the direct ratio of the antero-posterior and transverse enlargement of the lungs, but not precisely so of their vertical enlargement, in as much as there is no constant proportion between thoracic and diaphragmatic movement. The rapidity, the energy, and the extent of the expansion and elevation-motions, bear a direct proportion to each other under all circumstances of health, unless volition interfere to pervert the natural order of things. On the other hand the absolute amount of all three characters varies within sufficiently wide limits in different persons,—but is found to increase, as a rule, in the direct ratio of the easy mobility of the frame-work of the chest (hence greater in youth than age), and the height of the individual.

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* It seems probable that the state of relative upper and lower movement, in the male, is that designed by nature; the constraint of stays, in the female, interferes with expansion inferiorly; during sleep the condition in the female approximates, but does not coincide with, that in the waking male. For further observations on this point the reader is requested to refer to the Appendix.

During expiration the walls of the chest are restored to their previous condition by the converse movements of *retraction* and *depression*,—with greatest rapidity during the middle part of the act.

In each act of respiration the motions of expiration follow those of inspiration so closely, that no distinct pause is perceptible between them; when expansion and elevation cease, retraction and depression appear to begin, precisely as the audible pulmonary sounds by which they are accompanied.

If the entire time occupied by a respiratory act (that is, from the beginning of one inspiration to the beginning of the next) be represented by 10, the value of the duration of the inspiratory movement of the costal walls may be estimated approximatively at 5, of the expiratory at 4, and of the pause between the expiratory and succeeding inspiratory movement at 1: the period of thoracic motion is to that of rest, as 9 : 1. The movement at the close of inspiration is so small in amount, and so slowly effected, that it is very difficult to fix accurately the instant at which the actual rest, dividing any two succeeding respiratory acts, commences—hence, doubtless, the difference in the above ratio (which I have been led to adopt as the mean result of numerous observations) from that set down by some other writers.

In health the *extent* and *frequency of repetition* of the motions of the thorax are in the direct ratio of the *duration* and *intensity* of the pulmonary respiratory membranes.

(*b*) The *partial* motions, or those of the ribs on each other,—motions limited to special situations,—are best appreciated by application of the hand.

But the movements of the walls of the chest are not the only ones, dependent on respiration, which are of clinical importance. The enlargement of the lungs, and descent of the diaphragm in inspiration, forces down the subjacent viscera, and causes protrusion of the abdominal walls, especially anteriorly; during expiration the walls retract. Hence, in *ordinary* breathing, which is mainly affected by the descent of the arch of the diaphragm, the amount of abdominal, is greater than of thoracic expansion-movement, and the former commences sensibly before the latter: this is true, however, only of adult males; in females the costal expansion (especially superiorly) is greater than the abdominal—a difference, I presume to depend on the constraint of the lower chest and upper abdomen by stays, and other articles of dress.* In *forced* breathing, in both sexes, the thoracic movement is greater out of all proportion than the abdominal; and even in the male the expansile action commences superiorly. On these points further information will be found in the section on Mensuration.

MOTIONS, IN DISEASE.—(*a*) The general motions of simultaneous expansion and elevation are liable to *diminution*, either from instinctive avoidance of pain, paralysis of the muscles naturally producing the motions, or a material obstacle in the condition of the pleura or lung. Thus in pleurodynia, intercostal neuralgia, and the onset of pleurisy, the first cause acts; in cerebral and in spinal paralysis, the second; in the course of pneumo-thorax, pleurisy, pneumonia, obstruction of the main bronchus, gaseous, liquid or solid accumulations in the pleura, consolidation (tuberculous or other), and rarefaction of the lungs, the third. But these motions *exceed* the healthy standard, where a muscular effort is made to overcome some obstruction, seated lower in the chest, to the entry of air, as in spasmodic asthma, and especially if the diaphragm be mechanically interfered with, as in pericarditis with effusion. If, on the other hand, the upper air passages be obstructed, either from disease in themselves (simple œdema of the glottis, laryngitis, simple œdematous, or croupal, tumours, foreign bodies, &c.), or in neighbouring parts, (enlarged tonsils, pharyngeal tumours, &c.), or from spasm of the glottis, (as in epilepsy, hysteria, laryngismus stridulus, pertussis), and in chorea, the chest, instead of expanding during inspiration, will actually retract, and the abdomen enlarge, in proportion to the amount of obstruction. In many of these affections the rhythm of the movements becomes jerking and uneven; and according to their site they impair the expansion-movements, either bilaterally and equally, or unilaterally, or locally, only, over a limited portion of one side. Whenever any cause seriously impairs the expansion-movement of one side only, the expansion of the other is liable to increase; and similar deficiency of motion, limited to a part of one side, may be made up by excess on the rest of that side: the law is the same as for the audible phenomena of respiration. It holds good, too, where the obstruction is parietal, as in cancerous infiltration of the wall of either side of the thorax.

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* See Appendix.

Want of power in the respiratory muscles, whether from debility or from paralysis, will impair the chest-motions; in the tetanic spasm, whether morbid or from strychnia, the walls are fixed.

(*b*) The relation of the motion of expansion to that of elevation may change completely; the former, for example, being almost totally suppressed, while the latter becomes even peculiarly obvious. When the lung-substance is more or less impermeable, either locally or generally, and either from disease within itself or pressure from without, (as in cases of tubercle, pneumonia, pleurisy, pleuritic and pericardial adhesion, intra-thoracic tumour, aneurism, &c.), this kind of perversion in movement will exist, either locally or generally, according to its cause. It is especially marked on forced inspiration; volition may drag the thorax upwards, but cannot expand impermeable texture. In vesicular emphysema, while the elevation-movements are carried to an extreme point, there may be no elevation at all (or even slight retraction) at the base during inspiration.

(*c*) The *rhythm* of the *respiratory act* is likewise subject to change; the duration of the expiratory movement may become considerably greater than that of the inspiratory. This is observed wherever material obstruction exists, in any part of the passages from the nares downwards, to the exit of air from the lungs; and also (as in vesicular emphysema) where the elasticity of the lung is destroyed. In the latter affection the expiration-movement may be two-and-a-half times as long as the inspiratory.

(*d*) The proportion naturally existing between the extent and frequency of the *motion of elevation* on the one hand, and the duration and intensity of the *respiratory murmurs* on the other, may be altogether subverted; the former may be greatly increased in amount, while the latter have undergone remarkable diminution. This state of things constitutes one of the most remarkable features of diseases, where spasm affects the bronchial tubes or glottis, and obstructions of physical character exist in the larynx, trachea, or larger bronchi. The inspiratory action is abrupt and short, the expiratory prolonged.

(*e*) Again, the relationship of the thoracic and abdominal movements may change completely. Thus all conditions interfering, either dynamically or physically, with the movements of the diaphragm, while they impair these, give excess of energy to the thoracic class. Inflammation of the diaphragm, or of the nervous membranes coating it, great fluid effusion in the pericardium, solid, fluid, and gaseous accumulations in the abdomen, pervert in this manner the natural order of things. On the other hand, the diaphragmatic movements may be increased by certain irritations of the phrenic nerves, in pleurodynia, intercostal neuralgia, the painful periods of pleurisy, in diseases or injury of the spinal cord below the phrenic nerves, and (as compared with expansion-movement of the thorax) in obstructive diseases, functional or mechanical, of the air passages.

Before leaving the subject of inspection it may be as well to observe, that the undulation of fluid contained in the cavity of the pleura may be distinctly *seen* in some rare cases of considerable bulging of the intercostal spaces,—independently of perforation of the costal pleura, and escape of the fluid into the common cellular membrane. This is the only sign discovered by inspection which is not a modification of some natural condition.

SECTION II.—APPLICATION OF THE HAND.

BY application of the hand and palpation, are meant the acts of laying the hand on, and feeling, the external surface of the chest. The object of these acts is to ascertain the *form* of the different regions of the thorax (little or no information can be derived from them regarding the *general* conformation of the cavity); the state of the *general*, and especially of the *partial*, *motions* of the walls; the amount of *vibration* communicated under certain circumstances to the hand from those walls, and the existence or absence of *fluctuation* in the cavity of the pleura.

In employing this method of diagnosis, the palmar surface of the fingers and hand should be laid gently and evenly on the surface. If the object be to investigate the form or motions of the thorax, this is the only precaution, in addition to those recommended for the proper performance of inspection, which it is necessary to observe; if the thoracic vibration be the subject of examination, it is advisable to place the patient in the horizontal posture.

(*a.*) Application of the hand is less useful than inspection in ascertaining the amount of *general motion*, taken as a whole, existing in any given thorax; but it is greatly more effectual in distinguishing *locally expansion* from *elevation*-motion, and in analysing the *partial* costal movements.

Thus in chronic pleurisy with retracted side a good deal of elevation-motion may be felt during inspiration, while the total absence of any motion tending to fill out or expand the hand laid on the surface is readily ascertained. The same state of things may constantly be established in the infra-clavicular regions, when the apex of the lung is consolidated. In this case and also in empyema the thoracic walls, above and below the clavicle, may fall in during inspiration and expand during expiration, while the elevation-movement pursues its natural course and rhythm.

The sole means of accurately estimating the partial costal motions (those of the ribs in respect of each other) is by palpation. If the index-finger or thumb be placed in an intercostal space, anteriorly, of a sound chest, it will be found, that during inspiration the ribs diverge, and during expiration converge; the amount of divergence is obviously greatest in the lower interspaces, least in the middle ones. Allied in mechanism to the expansion-movements, these diverging-movements may nevertheless be affected differently from the former in disease. Thus, where the apex of the lung is tuberculised, the elevation-movement may, during inspiration, be still perceptible in the infra-clavicular region, and the expansion-movement be absolutely null, while the ribs actually converge. This inspiratory convergence of the ribs has appeared to me indicative of subjacent pulmonary consolidation and pleuritic *agglutination* combined.*

(*b.*) *Tactile vibration, or fremitus.*—If the hand be applied to the surface of the chest of a healthy individual, while speaking, a vibratile tremor is felt by the observer. This vibration, delicate under all circumstances and readily deadened by too forcible pressure of the hand, is, generally speaking, in the direct ratio of the graveness, coarseness of quality, and loudness of the speaking voice, and hence, as a rule, more marked in adults than in children, in males than in females.† It is often altogether deficient, indeed, in females, and habitually so in children. *Cæteris paribus* it is more intense in long-chested than in short-chested persons, and markedly so in thin than in fat people; unless as deepening the voice and either lessening or increasing fulness of person, age appears to have no influence upon its amount. The vibration is scarcely affected by tension or relaxation of the muscles over which the hand is laid; in the great majority of cases it is stronger in recumbency than in the sitting posture (in twenty-two trials, sixteen times greater lying than sitting, four times equal, twice more marked in the sitting than the lying posture). It is greatly more marked when some sounds are uttered than others, and hence the importance, in delicate comparative trials, of making the person examined repeat the same word or words.

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* Dr. Sibson (Med. Chir. Trans., vol. xxxi., p. 360) affirms that the five upper ribs converge during inspiration in the healthy state. I have never succeeded clinically in finding this, where the chest was sound, and very rarely where diseased. When a finger is placed in any upper intercostal space (the thinner the subject of the observation the better) it is compressed by the adjacent ribs during expiration, and relieved of all pressure during inspiration —just (though not to the same amount) as in the lower interspaces. *Vide* Appendix.

† In singing, the fremitus is much more marked when the voice is bass, baritone, or contralto, than when tenor or soprano; and it accompanies the

As a general truth, the intensity of the fremitus is considerably greater on the right side of the chest than the left,—the greatest amount of this excess existing in the infra-clavicular, infra-scapular, and inter-scapular regions. Exceptional regions are the right infra-axillary and infra-mammary, where the presence of the liver interrupts the vibrations, and throws into comparative prominence the naturally weak fremitus in the corresponding regions on the left side: the difference would be greater, were it not for the presence of the spleen in the latter position. Where the heart is uncovered by the lung, vibration is totally absent, and the right edge of that space may be traced by its abrupt cessation there; over the left lung there is naturally so little vocal vibration, that modifications of the sign can scarcely be used with confidence for making out the left edge. The lower border of the right lung may be traced by the abrupt cessation of all fremitus immediately below it.

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lower notes of any given register to a much greater amount than the upper; it may be absolutely null on a high note, though most loudly sounded, while it is well marked with a low note of the same voice softly uttered. From a few trials, I find that the fremitus ceases with soprano and mezzo-soprano voices between *f* and *a* on the lines; upper basses either retain the fremitus through their whole register, or lose it about their upper *f*.

The fremitus is intensely marked over the larynx and trachea, stronger at the sternal than the humeral halves of the infra-clavicular regions, generally faintly manifest on the right clavicle, and imperceptible at the top of the sternum.

In Disease.—(*a*) The natural fremitus produced by speaking is susceptible of *increase* or *diminution.* As in the case of other signs, the existing amount of change is most effectually ascertained by comparison of the two sides of the chest; but in making this comparison, the observer must carefully bear in mind the great differences naturally existing on these two sides. Unnatural density of the pulmonary texture, produced by infiltration, *unless this be carried to an extreme amount,* intensifies vocal vibration,—as in tuberculous or chronic plastic infiltration, and in acute hepatisation: pneumonia of the left base posteriorly will thus raise the fremitus above the standard of the right base in health. Pulmonary apoplexy and œdema act, within my experience, in the same way, but to a slight amount. In dilatation of the bronchi the increased calibre of the vibrating tubes, as well as adjacent consolidation, commonly tends to the same result. In pleuritic effusion occupying the lower part of the side, the infra-clavicular region may furnish fremitus in excess.

When the lung-substance is removed to a distance from the chest-wall by gaseous or liquid accumulation in the pleura (as in pneumo-thorax, in pleural effusions, dropsical, hæmorrhagic or inflammatory) the fremitus is destroyed. The influence of solid accumulation (either in the lung or pleura) varies according to circumstances: *very extensive* lung-infiltration, whether fibrino-plastic, pseudo-fibrous, carcinomatous or other, deadens the vibration, especially if the infiltrated parts be distant from the larger bronchi; extra-pulmonary tumors and aneurisms produce the same effect. But if the other circumstances be favourable (such as the tone of the voice,) aneurisms, tumors and cancerous infiltrations will not, even when of tolerable size, annihilate fremitus, if they be in close connection with the larger bronchi.

It is commonly said that in vesicular emphysema the vibration is impaired; I have not found this habitual, and in some cases its intensity is above the range of health.

(*b*) The act of coughing produces a vibration similar to, but less marked than the vocal. This vibration (*tussive*,) suffers the same kinds of modification in disease; but is valueless clinically, unless in cases of aphonia.

(*c*) Certain rhonchi throw the bronchial tubes into vibration sufficiently strong to be felt on the surface of the chest (*rhonchal fremitus*): the sibilant, sonorous, and mucous, have all this property. The cavernous rhoncus, produced in excavations of the lung near the surface, may be accompanied with marked fremitus, and without fluctuation being perceptible to the finger. Stridulous respiration even (such as that attending aneurismal pressure on the trachea) may produce very distinct fremitus, greater in inspiration than in expiration.

(*d*) In the natural state of the pleura, the gliding motion of its costal and pulmonary laminæ upon each other gives rise to no vibration perceptible by the hand applied to the surface. Nor is it usual, even in cases where *audible* friction-phenomena exist, to discover such vibration: in some instances, however, it may be detected; and the sensation conveyed, though distinctly somewhat vibratile, nevertheless possesses more of a simply rubbing character, just as might be anticipated from a consideration of its cause,—the friction of plastic matter on the pleural surfaces. In point of intensity it varies greatly; in some cases perceptible, even in ordinary breathing, to the patient himself,—in others it is only evolved by forced inspiration, and only to be caught occasionally. Accompanying either the inspiration or expiration-movement, it is more commonly associated with the former. I have met with it to a higher degree at the absorption-period, than at the outset, of pleurisy.

(*e*) A pulsatile movement of the lung, attended with a quasi-tremor on the surface of the chest, and synchronous with the systole of the heart, has been noticed (first by Dr. Graves) in some rare instances of pneumonia and intra-thoracic cancer. Here, too, may be included the impulse of pulsating empyema.

Palpation may also be used for the detection of fluids contained either in the lungs or pleura. The sensation is that of ordinary fluctuation, attended (not always) with a certain degree of vibratile tremor. Its existence may be ascertained, either by the movements of the fingers used by surgeons for detecting fluid in an abscess (*simple fluctuation*); or it may be necessary to perform succussion of the chest (*fluctuation by succussion*); or percuss the surface, in order to produce it (*"peripheric" fluctuation*); or it may occur through the influence of respiration as an attendant on cavernous rhonchi (*rhonchal fluctuation*); in this latter case it is that vibration may be most distinctly felt. The "peripheric" species (described by Dr. C. Tarral) is to be detected by giving a quick sharp fillip in an intercostal space, perpendicular to the surface, when a sensation of fluctuation will be transmitted to a finger of the other hand firmly applied to the surface in the same space, at a short distance from the point percussed. Simple fluctuation is producible, where the intercostal spaces are much bulged out by pleuritic effusion; peripheric fluctuation in the same cases, but most perfectly where air and fluid co-exist in the pleura; fluctuation by succussion in cases of hydro-pneumo-thorax and of large excavation in the lung.

SECTION III.—MENSURATION.

The object of measuring the chest is twofold: First, to ascertain, more accurately than can be done by inspection and application of the hand, the comparative bulk and volume of the two sides, the relative positions of their different parts, and, in some few instances, the distances between those parts and certain fixed points beyond the limits of the thorax (*measurements in rest*): Secondly, to estimate with precision the amount of expansion and retraction of the chest accompanying inspiration and expiration (*measurements in motion*).

A complete system of Mensuration would comprise the following admeasurements:—

A. GENERAL.—(*a*) Circular. 1. *On level of sixth cartilage;* 2. Midway between nipples and clavicles. (*b*) Transverse. 3. From point of one acromion to that of the other; 4. In axillæ; 5. At base of chest. (*c*) Antero-posterior. 6. *Under the clavicles;* 7. At base of chest. (*d*) Vertical. 8. *From clavicle to most dependant point of ribs.*

B. PARTIAL.—(*a*) Horizontal. 1. *From nipple to middle line of sternum.* (*b*) Vertical. 2. From middle of notch of sternum to nipple; 3. From nipple to antero-superior spine of the ileum; 4. From the most dependant point of the twelfth rib to the same spine.

But these different kinds of measurement are not all of equal importance, especially in the present state of knowledge,—either because some of them really convey information of very secondary value, or because they have not as yet been sufficiently practised to render the physician familiar with the indications derivable from them. The measurements which it is of real consequence for the student to understand, and in all doubtful cases to practise, are distinguished by italics: at the same time, it would be a mistake to imagine that all the others may not occasionally furnish more or less useful information, either confirmatory or corrective of results otherwise obtained.

§ I.—MENSURATION IN REST.

A. GENERAL MEASUREMENTS.—(*a*) *Circular,* (1.) *on the level of the sixth cartilage.*—Circular measurement of the chest, as commonly performed with a *single* graduated tape passed round the thorax from the middle line anteriorly, is a troublesome process, requiring the patient to be raised to the sitting posture, and the co-operation of two persons. Besides, the difficulty of ascertaining the precise point of the measure, corresponding to the spine, renders the process inaccurate. These difficulties have been removed by a very simple plan, suggested, I believe, by Dr. Hare,—that of joining together *two* such tapes at the commencement of their scales, and fixing them, as the patient reclines, at their line of union, to the spine: each side of the chest has thus its separate measure. By padding the inner surface of both measures, close to their line of junction, a sort of saddle is made, which readily fixes itself to the spinous processes.

The circular capacity of the chest varies so widely in healthy individuals, that there is little practical utility in attempting to fix its mean value. I have known it in the adult male so high, on the level of the sixth cartilage, as forty-four inches, and so low as twenty-eight. Perhaps thirty-three inches may be mentioned as the fairest adult average; but it varies with age (long after the height has become stationary), gradually increasing from the age of sixteen to sixty: so that the mean being thirty inches from the age of sixteen to twenty, it is thirty-four from that of fifty-one to sixty. The circumference increases, but not in any fixed proportion, with the robustness, stoutness, and height of individuals. The female circumference is, absolutely, less than the male.

According to M. Woillez, the circumference is greatest in persons following trades that require active exertion of the whole frame, but not of the upper extremities in particular. Far from this, were his number of cases sufficient for the final decision of the question, the latter class of artisans must be held to have the lowest average circumference of thorax. But the absolute measurement is of less importance than might appear, for width is not an index of expansile power; far from this, these statical and dynamical conditions may (as in fat people especially) be inversely as each other.

The relationship of pectoral to abdominal circumference varies with age. In infancy and childhood the latter is greater than the former; in the male adult (less so in the female) the chest exceeds the abdomen in width. In cases of abdominal obesity the natural ratio becomes perverted.

The two sides of the chest are of unequal semi-circumference in about five-sixths of healthy adults; a mean excess of about half an inch existing on the right side in right-handed indivduals;[*] in left-handed persons the left side sometimes measures more, or more frequently the same as, the right. These propositions hold true of both sexes; but the difference is slightly greater in males than females. In infancy and youth the two sides scarcely differ. I have not traced any special influence of trade on the measurements; but accidental circumstances (unconnected with disease of the thoracic organs) are liable to modify their ratio. Thus, distension of the stomach with flatus or food may equalise the measurement, for the time it lasts, on the two sides; and I am led to believe, from a case observed at University College Hospital, that the deficiency of motion, kept up by fractured ribs in some cases for a very lengthened period, perhaps for a life-time, may in the end diminish the dimensions of the side,—the lung probably (as in cases where its activity is impaired by bronchial pressure) becoming more or less atrophous, and the thoracic walls falling in proportionally.[†]

The most important *morbid conditions* of circular dimensions are *increase* or *diminution of width* of either side, as compared with the other. They occur respectively in the same diseases (already enumerated under these heads) as morbid expansion and retraction.

2. *Of circular width midway between the nipple and clavicle* little is known. The scapulæ prevent the real measures from being taken, and in some persons raise the circumference here above that on the level of the sixth cartilage; as far as is now

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[*] This excess becomes more remarkable when the frequency of slight convexity of the dorsal spine to the right, in health, is considered.

[†] Case of Bassett (Males, vol. ii. p. 214); the right side (the man being right-handed and free from pulmonary disease, capable of explaining the fact,) measured (opposite the sixth cartilage) $16\frac{7}{8}$ inches, the left $18\frac{3}{8}$ inches: he had old fractured ribs on the right side.

known, *the ratio of the two circumferences does not appear to* me sufficiently constant to be trusted to clinically. It is matter of general belief that the size of the upper part, compared with the base, is greater in proportion as the muscular and osseous systems, especially the latter, are strongly developed, and the constitution of the individual free from the taint of predisposition to phthisis. Though not prepared to say positively whether the excess of width, existing at the lower part of the chest on the right side, holds here also, I am inclined, from a limited number of observations, to believe that it does, though to a less amount than inferiorly.

(*b*) *Transverse.*—Respecting mensuration of the transverse diameters of the chest, I have no precise information to offer. It should be made with a pair of callipers, and there can be little question that diagnostic data of importance might occasionally be derived from its employment. It would, however, in the greater number of cases, do little more than confirm the results of inspection; for diminution of the transverse diameter, in respect of the antero-posterior, the change which, it may be presumed, would most frequently present itself, is one of those alterations of shape which most readily attract the eye.

(*c*) *Antero-posterior.*—A pair of common steel callipers is the simplest instrument for determining the antero-posterior diameter of the chest. The difficulties, in comparing the diameters of the two sides, are to apply the blades of the callipers with exactly the same force, and (what is far from easy, on account of the slope of the surface of the chest,) to exactly corresponding points on both sides.

In measuring the antero-posterior diameter of the apex of the chest on either side, the extremity of one blade should be placed immediately under the centre of the clavicle, the other upon the corresponding point of the spine of the scapula, the equi-distance of both extremities from the middle line being at the same time carefully ensured. The diameter of the right side in this situation (as also over the sixth rib) will be found, in the greater number of healthy persons, to exceed that of the left, but by so very small an amount that it need scarcely be taken into consideration where an excess at all marked is detected on that side. In other words, such an excess (for example, a fourth of an inch) on the right side, furnishes sufficient evidence of morbid depression or diminished diameter on the left; though the existence of a similar excess on the left will be still more strongly conclusive of contraction on the right.

The *morbid states* discoverable by the measurement now described are, *diminution* and *increase* of the antero-posterior diameter. The latter change occurs in pleurisy with effusion, pneumonia, hypertrophy of the lung, emphysema, intra-thoracic tumor and aneurism, various heart-affections, and possibly at the very outset of tuberculisation. Diminution, on the other hand, arises in the more advanced stages of tubercle, in simple chronic consolidation, and in the absorption-period of pleurisy with retraction.

(*d*) *Vertical.*—The vertical measurement of the chest has hitherto been only practised in front; measured with a tape, the distance between the centre of the clavicle and the most dependant point of the corresponding ribs is found to be the same on both sides.

This distance is liable to be *increased* in cases of solid, liquid, or gaseous accumulation in the chest; to be *diminished* in chronic pleurisy with retraction. But though elevation of the diaphragm, and consequent diminished vertical height of the thoracic *cavity* on either side, is common in cases of absorbed effusion, decrease of this measure on the *surface* is rare.

B. PARTIAL MEASUREMENTS.—(*a*) *Horizontal.* 1. *From nipple to middle line.* The nipples are equi-distant from the middle line in the healthy adult male. The distance between either of them and that line is liable in disease to increase and more frequently to diminution. Mediastinal tumors and aneurisms in certain situations increase it, though not often seriously; on the other hand, diminution in cases of retraction after pleurisy, varying in amount from a quarter of an inch to an inch and a quarter, may be detected.

(*b*) *Vertical.* 2. *From the middle of the notch of the sternum to the nipple.*—These points are equi-distant on the two sides in chests of perfectly regular form. We have already seen, however, that the left nipple sometimes lies naturally lower than the right; hence the space comprised between the nipple and clavicle on that side may be greater than on the other, independently of the influence of disease.

The only *morbid variation* observed in this measurement is *increase*, and it is obvious from what has just been said that this sign will have more value on the right than the left side. And mensuration is less useful in respect of this sign than inspection; for, on account of the flattening of the surface, which commonly coexists with lowered position of the nipple from disease, the superficial measurement undergoes a diminution which may more than compensate for the increase produced by the latter cause. In a remarkable example of this apparent contradiction between the results of inspection and mensuration, although the left nipple was manifestly lower than the right, the distance between the former and its corresponding sterno-clavicular joint was only 5½ inches, while that between the same points on the right side was 6¼ inches.* It is to be supposed that such will usually be the result of mensuration, where the retraction after pleurisy affects more especially the antero-posterior diameter.

3, 4. The distances comprised between the *nipple and the antero-superior spine of the ileum*, and between the *most dependant point of the twelfth rib and the same process*, are equal on the two sides in health. They undergo *diminution* on either side in cases of marked retraction of the chest; and, probably, *increase* in those of expansion.

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* Cyclopædia of Surgery, article Empyema, p. 102.

§ II.—MENSURATION IN MOTION.

There are two plans for submitting to measurement the influence of respiration on the dimensions of the chest; the one estimates the amount of antero-posterior movement, the other the amount of expansion and retraction attending the act.

(*a*) The former measurement is made by an instrument of very ingenious construction, named by its inventor, Dr. Sibson,* the "Chest-Measurer." In principle a callipers, of which one branch is moveable, this instrument is capable, by means of an index and dial fixed to its moveable branch, of indicating any change in the antero-posterior diameter of the chest or abdomen, even to the one-hundredth part of an inch, and has enabled Dr. Sibson to establish the following, among numerous other, propositions concerning the respiratory movements *in health*. In the robust male the forward movement of the sternum and of the seven upper ribs ranges from one-fifteenth to one-fourteenth of an inch in ordinary inspiration, and from half an inch to two inches during deep inspiration. On the five lower ribs the ordinary movement is less, and the forced movement greater, than over the upper seven. The movement is somewhat less on the left side than the right, below the second rib. The ordinary abdominal movement is about a quarter of an inch; the extreme ranging from about half an inch to an inch and a half.

Unless the expansibility of the chest be directly as the forward motion of the anterior parts of the ribs, the indications of the "Chest-Measurer" do not express accurately the amount of the former. When the costal cartilages are stiffened by age or precocious ossification, the expansion may be greater materially than in the ratio of forward costal motion; the converse state of things will exist in youth. In disease, too, the forward motion of the chest, and its lateral expansion, may be very differently affected: in a case of large excavation under the left, and consolidation with small excavations under the right clavicle, the lower part of the sternum and adjoining cartilages *receded* visibly during inspiration, yet a fair amount of *circular expansion* was produced by the act.* The deceptive influence of the torsion-movement of the ribs (which will apparently increase or decrease the amount of their forward motion, according as the moveable rack is fixed near their lower or upper edge) must be borne in mind; the very delicacy of the instrument might otherwise mar its utility. But in localising with precision deficiency or excess of antero-posterior motion, and in estimating the amount of either, the indications of the "Chest-Measurer" are greatly superior in perfection, it need scarcely be added, to those furnished by application of the hand.

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* Med. Chir. Trans. loc. cit; A modification of the instrument has been proposed by Dr. Quain.

(*b*) The amount of inspiratory expansion and expiratory retraction of the chest and abdomen is measurable by the double tape already mentioned. Applied closely, but not tightly, to the chest, on the level of the sixth cartilage, the tape shows that the expansion accompanying ordinary calm inspiration in health is very slight, averaging about a quarter of an inch in the healthy male adult, with a circumference of thirty-three inches: this amount is shared equally by the two sides. In forced inspiration the circumference is increased from the medium or tranquil state by from one-and-a-half to three inches, and is somewhat greater on the right than on the left side,—the deficiency on the latter mainly depending on the heart. The total circular difference between forced inspiration and forced expiration ranges between two-and-a-half and five inches: this is easily estimated by taking an admeasurement at the moments the patient has been made first to fill, and then to empty, the chest to the fullest possible amount. At the same time the difference in the respective extremes on the two sides is seen. But the same amount of difference may exist between extreme inspiration and extreme respiration on the two sides, and yet be very differently produced: it may, on one side, depend in the main on great expansion above, and on the other on great retraction below, the *medium state;* in other words, inspiration may be free to excess on the former, —expiration on the latter side. In health, however, such want of harmony in the expiratory and inspiratory efficiency on the two sides is never met with, except to the very slightest calculable amount, and is probably traceable to the difficulty of the observation.

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* Mary Green, F. Case Books, U. C. H. vol. v. July 1850. The same phenomenon sometimes occurs in pleuritic effusion also.

Forced breathing has scarcely any influence in expanding the abdomen: in a healthy male adult, five feet six inches in height, in whom extreme expiration gives a circumference opposite the sixth cartilage of 29½ inches, and extreme inspiration, one of 34 inches (a rare amount of respiratory play,) the abdominal inspiratory expansion equals only a quarter of an inch.

In disease, mensuration ascertains with accuracy the *amount* of deficiency of expansion on both sides, and on one side as compared with the other, the *mere existence* of which is more or less readily ascertained by the sight and touch. The Section on inspection may therefore be referred to for a list of the affections in which deficient expansion is to be estimated by measure.

The variations from the healthy standard thus discoverable are sufficiently striking. In chronic empyema, for instance, the total difference between the fullest expiration and the fullest inspiration may be scarcely one-sixteenth of an inch, while the other side (especially if time has elapsed for its lung to grow hypertrophous,) may have a play, as observed, of nearly two and a half inches,—an amount reaching the limits of health for both sides united. Hemiplegia will materially lower the respiratory play on the affected side: thus, in two cases elsewhere described,* the range of motion on the affected side equalled in each about a quarter of an inch, while that on the sound side measured three quarters of an inch and an inch.

When the respiration-play of both sides combined does not reach two and a half inches, disease impeding respiration in all probability exists: and when the total amount being equal to, or exceeding, a healthy average, the shares of the two sides are notably unequal, disease exists, certainly impairing the play of one side, and, it may be, exaggerating that of the other.

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* Clinical Lectures, *Lancet,* March 17, 1849.

But the measured range between forced expiration and forced inspiration may be the same on the two sides, and yet the accompanying change of volume of the two lungs be very different in kind. On the one side the play may be chiefly effected by the ready inspiratory expansion of the lung,—on the other, by the great efficiency of expiratory contraction. In the former case, the lung maintains its relative efficiency by its power of taking in beyond its medium quantity of air: in the latter by its power of expressing air, which in ordinary breathing stagnates within it; in the former case expansibility, in the latter elasticity, predominates. Here are two very different conditions of lungs, most important to be distinguished, and which have hitherto never been made the subject of *clinical* study. The difficulty of the study is, it is true, extreme, from the nicety required in fixing the standard of comparison, namely, the medium or tranquil measurement of the chest. My observations on this matter are as yet too limited to justify me in announcing general conclusions; but they prove to me that with care the inquiry may become rich in results.

The *forced* breathing of health expands the chest out of all proportion with the abdomen: in disease seriously affecting thoracic expansion, the diaphragm assumes unusual energy, descends more than natural and expands the abdomen. Severe pleurodynia will suffice to transpose the respiration-movements in this way. Where local rheumatism affects the parietes of the abdomen and chest both, it is curious to observe the medium state of modification in the relationship of abdominal and thoracic expansion in forced breathing: the pectoral expansion is relatively somewhat less, the abdominal somewhat more, than the healthy average.*

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* Case of William Farrett; U. C. H. Males, vol. vi., Nov. 1850.

(*c*) The expansibility of the lungs may be estimated in another way,—by measuring the cubic inches of air which can be expelled from the chest by the fullest possible expiration, succeeding the fullest possible inspiration. The most accurate investigations of this kind are those of Dr. Hutchinson, made by means of his valuable instrument, the Spirometer. These investigations appear to have given a satisfactory clue to the apparently absurd discrepancies of previous observers on the maximum volume of air which can be expelled from the chest; for they have shown that the bulk of air, receivable by the lungs in health, increases in a certain calculable ratio with the height of the individual: that (the mean volume being 174 cubic inches for the height of five feet) for every inch of stature, from five feet to six feet, eight additional cubic inches of air at 60° Fah. are given out by a forced expiration. Although numerous exceptions are encountered to this important law, there can be no doubt that it closely approximates to the truth in the main.

A *general* standard of health being admitted to exist, the question arises, what amount of deficiency below that standard actually indicates disease. It is said that a deficiency of 16 per 100 is suspicious, but may possibly arise from physiological peculiarity,—that beyond this, the deficiency is morbid.

But in practice it turns out, that the general standard of height is often valueless,—that the *individual* healthy standard occasionally varies widely on either side of the general one. So much so, that a great fall may have taken place, from disease, in the breathing-volume of an individual, at a time when he expels a quantity of air above the average standard of men of his height: according to the general standard he is more than healthy, he is extra-capacious; according to his own, he is diseased. For certainty of observation, the *individual* standard is required: the present man must be compared with the past man, and not with other men. Again, fall below the general average, is a surer indication of disease than the maintenance of the general average, or even a slight excess, is of health.

But of what disease? Obviously of any disease, whether situated in the lungs and appendages, the heart and great vessels, the abdomen, the encephalon or cord, which interferes on vital or mechanical principles with the expansion or retraction of the lungs. The spirometer indicates imperfect expansibility of the lung, but neither marks the seat nor the nature of its cause, unless observation should prove (and of this there seems no present probability) that special scales of reduction of capacity obtain in particular diseases. In this point of view (as well as, of course, in indicating which side of the chest is solely or most diseased) Spirometry appears to me inferior to semicircular mensuration of the chest.

SECTION IV.—PERCUSSION.

The act of striking the external surface of the chest for purposes of diagnosis is called *percussion;* and the immediate object of the process is the determination of the density of subjacent parts. Applied to the thorax, it serves to establish, by inference, any increase or diminution of the quantity of air naturally contained within that cavity.

The amount of density is inferred from, (*a*) The nature of the *sound* elicited by percussion; (*b*) The *degree of resistance;* in other words, the elasticity, of the body percussed.

(*a*) *Sound.*—The properties of percussion-sound, which, varying with the density (and some other physical conditions) of the textures and materials furnishing it, possess practical importance, are:—*Its degree of clearness; its duration;* and *its quality.*

Clearness.—The conditions of percussion-sound commonly described as *clearness,* and its converse, *dulness,* are scarcely capable of being described: they are readily illustrated by percussing, successively, the chest, at its antero-superior part, and the thigh; and the sounds elicited in these two situations, the former *clear,* the latter *dull,* may be used as measures of comparison for the greater number of sounds producible in various parts of the thorax. Although obviously incorrect, the terms *dull* and *clear* are retained in this work, because, in the first place, their practical signification is generally understood; and, in the second, it is extremely difficult, if not actually impossible, to substitute correct scientific expressions for them. They are incorrect; because *dulness* and *clearness* are not terms opposed to each other, either in the common signification of the words, or in an acoustic sense: nor are dulness and clearness * admitted among the properties of sound by natural philosophers; and hence there is this curious contradiction in the works of those writers on physical diagnosis who preface their volumes with an inquiry into the theory of sound, that no such properties as dulness or clearness are ascribed to it, and yet dull and clear sounds are perpetually spoken of in subsequent descriptions. Again, dull sound is used as synonymous with "little" sound, or "no" sound. This is sometimes, but not always, correct; for there is, in point of fact, as intense noise in many so-called dull, as clear, sounds. It is not in *intensity* that the difference which impresses the ear consists, but in *duration* and in pitch: so long as they both last, one is as intense as the other. Hence it would appear that what is practically called clearness signifies continuousness of sound; and dulness, non-continuousness. And as continuousness of sound depends on the elastic, vibratile character of the material furnishing it, the inference follows that when dulness exists, the material struck is either inelastic, or all vibration is suddenly stopped by some extrinsic influence. In clear and dull sounds, too, there is this further difference,—the former, as those of the chest, approach in character to tones; the latter, as of the thigh, are (at least as transmitted to the ear through the air) utterly toneless and mere noises.

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* The word clear applied to sounds, strictly speaking, means pure. The notes of an instrument are said to be clear, when they are heard singly and purely, uninterfered with by any extraneous vibrations;—the tones of the human voice, when free from huskiness or any other superadded character impairing their singleness. Now, in this sense no sound elicited from the chest by percussion can be called clear.

Under certain circumstances it becomes possible, even, to assign rudely the pitch of the percussion-sounds of the chest,—they become notes. This occurs more especially when, in addition to other conditions, the ribs are peculiarly favourable, from form or texture, to vibration. When the percussion-sounds are listened to directly through a solid stethoscope applied to the surface struck, the difference of pitch on different parts of the chest becomes more obvious than when the sounds are heard through the air.

All wet animal textures in a state of relaxation (with the exception of bone and cartilage, which possess a special clear resonance,) furnish a dull sound, or rather a mere noise, under percussion. The viscera are indeed, practically speaking, soundless in themselves,—the proper substances of the liver, spleen, kidney, heart, and lung (from which the air has been artificially expressed), do not appreciably differ in regard of this property: all of them are deficient in the molecular elasticity required for continuous sonorousness. Hence the resonance of the lungs, of which we speak clinically, depends not on their proper tissue, but on the air they contain, and on the construction of the case in which they are contained. The quantity of bone and cartilage entering into the composition of that case, its hollowness, and the thinness of its walls, in comparison with the extent of its cavity, all conduce to the freedom of sonorous vibration. So, again, equal portions of heart-substance and of liver-substance, when similarly percussed, will give out sounds short, abrupt, and toneless, in no wise distinguishable from each other; yet the heart and liver, *in situ*, sound differently: the pitch of the heart-sound is perceptibly higher than that of the liver, and the difference depends on the hollow form of the former, *plus* the different properties of the cavities containing the two organs.

Duration.—Difference of the duration of the sounds emitted by bodies of different kinds under percussion may be illustrated —if examples of the familiar fact be required—by the prolonged ringing sound produced by striking a gong, and the short abrupt one similarly yielded by a mass of putty. The disparity in these two instances is considerably greater than any observable in percussing the human body, but less degrees can readily be conceived: that existing between the sounds emitted by the thigh and the cranium exemplifies one of those degrees.

The duration of the percussion-sound varies also very distinctly in different parts of the chest; for instance, at the upper part of the sternum and over the heart. The greater the dulness, the shorter (we have seen) is the duration of the sound; but as changes in the former are much more readily appreciated than in the latter property, this is not one from which much information is derived in practice.

Quality.—The quality (that property which essentially distinguishes any variety of sound from all others) of the sound emitted by the chest in health is not easily described; the usual statement, that it is a "good clear" sound, manifestly gives no distinct notion of its nature. It conveys the ideas of softness and of hollowness to a moderate degree; but is in fact *sui generis*, and a few trials upon a healthy chest will make the student more familiar with it, than could the most laboured description. The healthy quality (which, for brevity's sake, may be called *pulmonary*) is sufficiently marked and peculiar, to render the variations to which it is subject in disease easily perceived.

(*b*) *Degree of Resistance.*—When percussing a chest perfectly free from all disease, the observer is conscious of a slight yielding motion on the part of the walls, accompanied with a sensation of elasticity. It is impossible to fix the degree of this elasticity, but the reality of its existence may at once be ascertained by percussing comparatively the anterior part of the thorax and the thigh; in the latter situation a sensation of dead unyielding resistance is experienced. The amount of resistance varies inversely as the clearness of the percussion-sound; and directly as the amount of bone (ribs) in the walls.

Considered in respect of the manner of performing it, percussion is either *immediate* or *mediate*.

Immediate percussion, the invention of Avenbrugger, is performed by striking the surface of the chest with the points of the four fingers of the right hand, united into a point on a level with each other, the ball of the thumb being placed firmly against the index finger opposite the articulation of the second with the third phalanx, so as to support and give firmness to the fingers. The hand being thus prepared, the points of the fingers are brought perpendicularly down upon the surface with a sharp and quick stroke, which is found to produce a sound varying in properties with the condition of the subjacent parts. Avenbrugger recommended, as an important precaution, that the patient's chest should be covered with a thin dress, or that the observer should wear a glove,—the object being, by either plan, to prevent the sort of clack resulting from the contact of the naked hand and skin. If, in accordance with the advice of Laennec, the hand be kept naked and the chest covered, it is very necessary, as pointed out by Avenbrugger and others, that the shirt or other covering be drawn tight over the part percussed. Immediate percussion may also be performed by striking the chest with the palmar surface of the fingers: others tap the surface lightly with the distal end of the stethoscope; but patients always dislike this, and it may be productive of serious pain.

Immediate percussion has, however, almost completely fallen into disuse, less in consequence of the positive objections to its employment than of the discovery, in mediate percussion, of a plan, if not more ready in its application, much more satisfactory in its results. And, in truth, those objections are, in themselves, far from unimportant. Direct percussion, even when very skilfully performed, rarely fails to give more or less pain: in the hands of an awkward and inexperienced person it is really almost unbearable, and may, with some colour of justice, —because a more efficacious and less painful method can be employed,—be regarded as a piece of cruelty to the patient. Direct percussion cannot be performed over the intercostal spaces; and when the patient is very fat, or the subcutaneous tissues are anasarcous or emphysematous, its results cannot at all be depended upon. Besides, the manual process of immediate percussion is exceedingly difficult of acquirement, and the least inattention to the manner of its performance will scarcely fail of leading to erroneous notions of the state of the chest. Mediate percussion is, no doubt, liable to a certain extent to the same objection, so much as to make its practice much more difficult of attainment than that of auscultation; but there can be no doubt that the chances of error are with it much less numerous and serious. There are, however, a few circumstances under which immediate percussion may still be had recourse to with advantage. Thus, in cases where extensive and notable difference between the two sides exists, rapidly striking them with the palmar surface of the hand will leave no doubt as to the fact; indeed it will disclose the amount, though not the superficial extent, of the alteration of sound, almost as satisfactorily as the more delicate process of mediate percussion. In cases of hepatisation and of pleuritic effusion, where it may be inconvenient to submit the patient to a lengthened examination, this method, therefore, has its utility. Again, it will be found that directly tapping the clavicles and spines of the scapulæ with the points of one or more fingers, or with the bent knuckle of the index-finger, conveys as correct information as mediate percussion of those parts.

Mediate Percussion.—The distinctive character of mediate percussion, for the invention of which we are indebted to M. Piorry, is that some solid body, interposed between the chest and percussing agent, receives the *direct* impulse of this. In mediate percussion (or, as I shall in future call it, simply, percussion) there are two chief things to be considered—the material interposed, and the agent used for striking it.

The material interposed, termed a pleximeter, (πλῆξις, percussion, and μέτρον, a measure,) may be of different kinds. That employed by M. Piorry is a thin, circular, or oval plate of ivory, about an inch and a half in diameter, and provided with two prominences or handles, fixed at right angles to its plane surface, and at nearly opposite points of its circumference; these enable the observer to hold it steadily, and apply it evenly and firmly to the surface. Innumerable have been the modifications of this, and the varieties of new pleximeters, proposed from time to time; of these the left index finger (Skerrett?), and a flat piece of india-rubber (Louis), are in my mind decidedly the best. The index or middle finger, on account of their always being within reach, on account of the accuracy with which they may be fitted, as it were, to the various depressions on the surface, and on account of the absence of parade in their employment, will no doubt always continue the pleximeter in most common use. They have, in these points of view, an unquestionable superiority over M. Piorry's plate of ivory. The india-rubber pleximeter may, however, be defended: there is nothing pompous in its appearance, and by a little management it may be accurately applied, even in the intercostal spaces of the thinnest persons. It has, besides, this positive advantage, that it saves the finger of the operator,—no trifling matter, where a very large number of patients are to be examined. And its use implies a saving of pain not only to the operator, but also to the patient, as I ascertained some years ago by a considerable series of comparative trials. Some individuals bear percussion without murmur in this way, who resolutely refuse to allow it, if the finger be used for a pleximeter. The only objection I have ever heard urged against the india-rubber is, that it deadens the sound. This, which would be a valid argument if a single point only of the chest were to be percussed, and a direct inference drawn from the result, has in reality not a particle of force; because inferences are invariably drawn from the *comparison* of different parts.

Whatever pleximeter be employed, it should be placed in accurate and firm contact with the surface; for this reason it appears advisable to apply the palmar, and not the dorsal, surface of the finger to the chest, when this is the pleximeter used. No extrinsic condition modifies the sound so much as the amount of force with which the pleximeter is applied to the surface; and the finger with its dorsal surface turned to the chest is, in this point of view, comparatively unmanageable. The force of this objection is, however, not generally felt; M. Louis, among others, very frequently percusses in this way, and Dr. Stokes appears to prefer it. It is certainly, in some cases, easier to apply the dorsal than the palmàr surface of the finger *uniformly* to the part of the chest under examination; but this advantage has always seemed to me much more than counterbalanced by the disadvantage just insisted on.

The finger may be applied parallel to the ribs, or at various angles with them. The former way of placing it is infinitely the more common; and, as a general rule, is by far the more correct, for by it only can the finger be fitted, in thin persons especially, to the irregularities of the surface. But it is sometimes both convenient and advantageous to vary the direction of the finger; and, as it is next to impossible to place the finger uniformly and equably against the surface in the neighbourhood of the right acromial angle, if it be applied horizontally, fixing it at a variable angle with the ribs becomes a matter of necessity. To obviate the difficulty referred to, some persons stand *behind* the patient while percussing the upper anterior regions: but when this plan is followed, it becomes as difficult to fix the finger on the *left* side, as on the *right* when the physician stands in the usual way in front of the patient; the position is besides open to several other manifest objections.

Useful information may sometimes be obtained by using the four fingers of the left hand, laid firmly and closely on the surface, as a pleximeter. When the anatomical cause of variation of sound is considerable in extent, but slight in degree, there is an obvious advantage in including a space of some size under the pleximeter.

Whatever be the pleximeter used, the fingers are commonly employed as the percussing agent. The various hammers and accompanying apparatus, invented in this country and abroad, some of them of an appearance to terrify a timid patient, do not seem to me to possess any kind of superiority to the fingers, and labour under the serious disadvantage of depriving the observer of the indications furnished by the sensation of resistance of the parts percussed. The clinical supremacy of the fingers has never been a moment threatened by any of these elaborate inventions; and it appears consequently unnecessary to describe them. However, some practised observers prefer the hammer; and, on the principle of *audi alteram partem*, I would refer the reader to a valuable paper by Dr. Hughes Bennett in the "Edinburgh Monthly Journal," Oct., 1850. Generally speaking, the index and median fingers, having their points placed upon exactly the same level, and supported by the thumb with its ball laid firmly upon the outer surface of the former, opposite the articulation of its second and third phalanges, make the best instrument for striking with. But the index-finger alone may be used, especially when gentle percussion only is required, and, generally, therefore in the case of children. Under some circumstances three fingers form a useful modification; or the knuckle of the index-finger (joint of first and second phalanges) may be used with good effect; in percussing the larynx, the most convenient plan is to fillip with the median finger.

When the four fingers on the left hand are used as the pleximeter, those of the right form the best agent for percussing with,—the palmar surface of the latter (held in firm extension) striking the dorsal of the former.

In the case last referred to, the percussing fingers are made to fall *horizontally* (the more accurately so the better) upon the surface struck; under all other circumstances, it is of essential importance that the points of the fingers fall *perpendicularly* upon the pleximeter. The least variation in this respect is liable to be attended with a difference in the sound elicited.

In the act of percussing, *the movement should spring from the* *wrist only*, the fore-arm and arm being held perfectly motionless. The pain which beginners cause the patient in many cases, and the uncertainty of the results obtained, in a great degree depend upon ignorance of the value, or neglect, of this rule: the awkwardness of striking from the elbow, or even the shoulder, as is often done, is a matter of less moment; though an observant patient will scarcely fail to be impressed unfavourably by it, when he finds himself rather pushed about than percussed. But the essential advantages of this mode of percussing are the nicety with which the force of the blow may be regulated, and hence made precisely equal in any two places it is the object to compare; and the great comparative ease of keeping the percussing fingers at *the same angle* in striking repeatedly the same or different spots. Were this point of manipulation generally attended to, it would be infinitely less common, than it now is, to hear a new and different sound elicited by each of a number of successive blows upon the same place;—a variation, the mere possibility of which constitutes a serious drawback to the utility of percussion, as it is too frequently practised.

The force used in striking should never be great, absolutely considered; but it may be made to vary from the most gentle, to a smart tap, according to the object in view. Generally speaking, gentle percussion is advisable, when we desire to ascertain the amount of density of superficial parts; forcible, when deep-seated tissues are the subject of investigation. Corresponding regions of the chest, which yield sounds of the same clearness and duration when gently struck, may yield sounds materially differing in these respects if forcibly percussed, and *vice versâ*; it is therefore obvious, that both modes should be employed in every instance where accuracy of diagnosis is aimed at.

The blow should be *quickly* and *lightly* given, the fingers being withdrawn, or at least all pressure removed, the moment their impulse has been effectually communicated to the surface struck; the vibrations of the surface are thus impeded to the least possible amount. To this precept there is but one exception: in eliciting a particular modification of special character of the sound (*cracked-metal character*), the successful production of which depends materially on the manner of striking, it is advisable to give the impulse slowly and heavily, and allow the fingers to press forcibly on the part for some moments, after it has been given.

The posture of a patient undergoing percussion should, unless special circumstances prevent it, be the sitting or the standing. This precept is in accordance with the rules of Laennec, but perhaps his motives for laying it down were not perfectly well grounded. I cannot say I have found, as he represents to be the case, that "if the patient be in bed, the matrass, still more the pillows, and also thick curtains, always render the sound less;" and even admitting the fact to be so, as the object is to obtain comparative and not absolute results, it would constitute an objection of but slight importance. The difficulty of placing the patient perfectly level in bed (and if he be not so placed, the sound on either side is extremely liable to be modified), together with the constrained positions the physician is obliged to place himself in, in order to get at different parts of the chest, appear to me to constitute more solid objections to the recumbent posture.

While the anterior regions are under examination, the patient must hold his head erect and allow his arms to hang loosely by his sides; his hands may be clasped across the head, to facilitate percussion of the lateral regions; and he should cross his arms pretty tightly in front, and bend his head slightly forwards, while the back is examined.

Where muscle of any thickness covers the part examined, it should be in a relaxed state, so as to facilitate as far as possible the close approximation of the pleximeter to the proper wall of the chest. The converse is the case when immediate percussion is employed; for the obvious reason, that a flaccid mass of muscle, in itself scarcely vibratile, must, besides, interfere with the transmission of sound from the subjacent parts.

It is scarcely necessary to insist upon the importance of observing, as far as possible, the same conditions, when percussing the two sides of the chest comparatively. Nor must it be forgotten, that in doubtful cases, the observation should be repeated many times and in various postures,—more especially, if the patient be in bed, the percussion should be performed several times from the right and left sides of the bed alternately. On the other hand, it is of essential consequence in some cases (for instance in percussing the heart) that not only the posture of the trunk be unchanged during examination, but that the limbs be kept perfectly quiet.

Results of Percussion in the natural state.—It is difficult, as has been said, to make intelligible by words the *quality* of the sound elicited from a healthy thorax; experience only can teach it. The *duration* and the *clearness* of the sound bear a definite relation to each other; whenever the former is considerable, the latter is proportionally marked, and *vice versâ*. And again, the clearness of the sound and the sense of resistance experienced by the fingers have a manifest connection: as the former increases, the latter decreases. Thus the sound is clearer in the infra-clavicular region than in the scapular; and so the sense of resistance is much less under the clavicle than upon the scapula. It is true there are a few exceptions to this law of relationship of the clearness of the percussion-sound to the resistance of the percussion-surface; instead of invalidating, however, they strengthen the rule. Thus in the internal division of the clavicular and in the upper-sternal regions, the sound is clearer than in others—for example, the infra-clavicular —where the resistance is less. This peculiarity manifestly depends upon the nature of the wall of the thorax in the former situations: being there wholly composed of bone, it cannot give way and rebound under percussion to the amount which the slight density of the subjacent parts would otherwise ensure; while, on the other hand, bone possesses a special resonance comparatively clear and high-pitched.

✓ The properties of the percussion-sound of the healthy thorax vary materially in its different regions.

(*a*) *Anterior Regions.*—It is necessary to assume a standard of comparison for the sounds in different localities, and that of the infra-clavicular regions may be used for this purpose. Here the sound is of considerable clearness, true pulmonary quality, sufficiently prolonged to have a distinctly appreciable duration, while the parietal resistance is slight, and the elasticity marked. In the right mammary region, even at the upper edge, the clearness diminishes slightly on pretty firm percussion; while at and below the fourth interspace (though pulmonary resonance may be elicited by a very gentle tap) very perceptible dulness is caused, if the blow be firm, by the presence of the liver behind the shelving border of the lung: at the sternal edge of this region the heart in the majority of persons deadens the sound. In the inner part of the corresponding left region, the heart lessens the clearness of the sound and increases the resistance: this influence may be perceived more or less obviously as far outwards as the nipple. The entire of the infra-mammary region is dull and resistant from the presence of the liver: the left lobe of this organ habitually renders the inner part of the left corresponding region dull; while the outer portion is similarly affected by the spleen, and the intermediate space gives an amphoric and ringing or tympanitic note from the subjacent stomach, if empty,—a dull one, if full. On full inspiration a certain extent of lung encroaches in front of the liver superiorly, and may be discovered by very gentle percussion; even in expiration some lung-substance may be similarly detected in the left region, unless the stomach happens to be greatly distended.

Both clavicles towards their sternal ends give a peculiar sound of mixed pulmonary and osteal character, rendered somewhat tubular too by the immediate vicinity of the trachea; about the centre of the bone the tubular and osteal characters become less, the pulmonary more, marked,—whence an appearance of less clearness in the sound. Towards the humeral end of the bone the sound loses considerably in clearness. In the supra-clavicular regions externally (especially in females) the sound is quite as clear as in the infra-clavicular; clearer in the inner part, where a slightly ringing character is also elicited, unless the percussion be directed so as to avoid the trachea completely. The full resonance of this region may in moderately thin persons be detected even a little behind the anterior edge of the trapezius muscle. On the other hand, in persons free from thoracic disease, but either naturally very thin or emaciated from any disease, the outer part of this region often gives a wooden sound under percussion, and is very resistant; the first rib forms, as it were, the floor of the region, and its proper osteal resonance comes out modified by the intervening soft parts.

The supra-sternal region gives distinctly tubular resonance, if the percussion be made at right angles with the surface; a mixture of osteal sound is detected, if the impulse be given even to a slight degree in a downward direction,—the proximity of the trachea and sternum explains this. In the upper sternal region the resonance is of the same mixed quality, as on the adjoining end of the clavicle, as far downwards as the second rib: here, at the point of convergence of the inner borders of the lungs, (especially at the moment the organs are distended by inspiration,) purer pulmonary sound may be elicited by gentle percussion. But occasionally, from excess of cellulo-fatty substance at the top of the mediastinum, the percussion-sound may be perfectly dull here,—a peculiarity which sometimes proves a source of serious clinical difficulty.*

From the third rib downwards the resonance is of complex character. The heart and liver with the intervening diaphragm give a dull sound, modified, however, by the peculiar bone-resonance of the sternum itself. If the stomach be in the condition to yield either an amphoric or a tympanitic note, one or other of these qualities may be detected, modifying still further the dull sound of the solid organs. This region further presents one of the most difficult practical problems in the art of percussion,—that of defining the line of union of the heart and liver. Now, although there are cases where to a practised ear and finger the differences in the pitch of the sound and resistance of the two organs will effectually mark out the contiguous edge of each of them, instances of the kind are rare. These characters failing, the line of demarcation may in many persons be traced by making gentle and forcible percussion alternately, while the patient holds his mouth widely open: the special amphoric quality of the stomach-note may then be detected between—and only between—these two organs; this I have repeatedly proved on the dead subject. But even this test will, sometimes from solid or fluid accumulation in the stomach—sometimes from unusual position of the viscera, wholly fail; the line can then only be found by uniting hypothetically the lowest point of the heart's impulse with the apex of the angle formed by the union of the upper free edge of the liver with the right free edge of the heart.

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* It may simulate, according to circumstances, the appearances of pericardial effusion, aneurism or tumor,—as will hereafter be fully shown.

(*b*) *Posterior Regions.*—The sound yielded by the upper scapular region, though less clear than that of the infra-clavicular, is not, absolutely speaking, dull, nor altogether without pulmonary quality, even in fat persons; in very thin people the sound is sometimes very notably clear. The lower scapular region, while duller than the upper, and resisting to a marked degree the impulse of the fingers, has some faint pulmonary character in both respects; the spine of the bone, on mediate percussion especially, gives a clear osteal sound. The interscapular region holds an intermediate place in point of resonance between the upper and lower scapular.

The entire of both infra-scapular regions, especially in inspiration, gives a clear pulmonary note on gentle percussion as far as the eleventh interspace downwards. But forcible percussion on the right side brings out the dull hepatic sound from the lower edge of the region, as high as the ninth or even eighth rib, the amount of dulness gradually decreasing in the upward direction. On the left side the spleen, if large, or the stomach and intestines, modify the percussion-sound of the lung at the lower part of the region. Speaking roughly, it may be said that the extreme right base sounds somewhat duller, posteriorly, than the left. The influence of the kidneys, when of natural size, is null.

(c) *Lateral Regions.*—The axillary region is highly resonant in its upper part, giving in many persons a sound clearer and of greater duration than the infra-clavicular region. On the right side the sound becomes dullish on strong percussion at the fourth interspace, and quite dull at the sixth rib, continuing so thenceforth to the lower border of the infra-axillary region. Below the sixth rib on the left side, though pulmonary resonance may still be elicited by gentle percussion, the spleen and stomach modify in their respective ways the quality of the sound. Lastly, the sound elicited from the larynx and trachea (which is best obtained by filliping with the right middle finger one of the fingers of the left hand applied firmly to the surface, the patient's head being thrown back, and the tissues of the neck thus brought into a state of tension) is very clear, of considerable duration, and has a distinct hollowness in its quality,—is in fact tubular. These characters arise from the form of the part and the quantity of cartilage in its walls. The resistance is greater under the fingers, in proportion to the clearness of the sound, than it would be in the chest.

The properties of the sound elicited by gentle and by forcible percussion differ to a slight degree in all regions of the chest. When strong percussion is used, the percussing fingers, by a sort of mechanical necessity, are allowed to rest on the surface for a moment; and thus a muffled character is given to the sound from interference with the vibrations of the chest-walls. Hence, force in the blow, instead of intensifying, weakens the sound and lessens its duration. Again, wherever an organ of greater density than the lung lies at some depth from the surface, the intervening space being occupied by pulmonary tissue, the sound will be rendered duller by striking heavily, its duration diminished, and the sense of resistance increased: by employing force, the impulse is made to reach the deep-seated organ. For this reason a decrease in clearness may, as we have seen, be sooner detected by strong than by gentle percussion, in passing from above downwards in the right lateral and anterior regions; the influence of the liver is thus brought into play at a higher point of the chest. On the same principle the precise extent of the heart overlapped by the lung may be defined by alternately using some force, and by merely tapping the surface.

Cæteris paribus, the sound is clearer on the ribs than the intercostal spaces; this is especially true in thin persons,—the osteal sound mixes with the pulmonary, and raises the pitch of the resulting compound sound.

Numerous differences in the results of percussion in corresponding points of the two sides of the chest have been referred to and traced to an obvious cause,—the presence of texture and organs of different densities in those spots. Variable thickness of the external soft parts will have a similar effect; thus the right infra-clavicular region is less resonant than the left in robust persons whose employment requires much use of the right arm: the pectoralis muscle enlarges from use. Here the explanation is clear; but the explanation is not clear when the right infra-clavicular region sounds duller than its fellow, in persons presenting no muscular thickening of this kind.* Such inferiority of clearness is, however, the lungs being perfectly sound, always when it exists, very slight: it holds good, whatever be the direction given to the percussing fingers, and whether they fall outwards towards the humerus or inwards towards the sternum.

The acts of inspiration and expiration modify the results of pulmonary percussion in three different manners: 1. by altering the volume of the lungs; 2. by altering their density; 3. by altering the position of the heart and abdominal viscera.

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* Clinical Lectures, loc. cit. p. 196.

1. At the close of an ordinary expiration the right lung extends downwards as far as the sixth rib in front and the eighth laterally. The lower edge of the left organ sloping abruptly outwards and downwards from the middle line on the level of the fourth cartilage, again turns inwards a little, and then passes outwards, reaching laterally the eighth rib or interspace. From numerous trials on the dead subject, when no cause existed to modify materially the volume of the lungs, I infer that in expiration the lungs are from one to one and a half inches apart on the level of the second cartilage, and four and a half inches apart on the level of the fifth. From their lateral aspect the lower edges curve downwards and backwards, reaching the ninth interspace or tenth rib, especially close to the spine.

During full inspiration the lungs extend downwards in all directions somewhat further than the limits just mentioned; their inferior edge is then carried about an intercostal space and a half lower than after ordinary expiration, and proportionally still lower, when the lungs had been forcibly emptied of their air. At the same time the space on the left of the sternum, where, after expiration, the heart is in contact with the thoracic walls, becomes filled with lung.

It is obvious, from these facts, that the *superficial extent* of surface, from which pulmonary percussion-sound may be elicited, will vary with the precise moment of the respiratory act at which the observation is made.

2. The density of the pulmonary tissue being in the inverse ratio of the air it contains, it is plain that the percussion-sound must be rendered duller in any given point of the chest by a full expiration. In other words, the sound yielded will vary in clearness according as percussion is performed at the close of inspiration or of expiration. The duration of the sound, and the sensation of elasticity perceived, vary, in the ordinary way, as the modifications of clearness.

3. The depression of the dull-sounding spleen and liver attending inspiration, replaced as they are by expanding lung, tends during that act to increase the clearness of the percussion-sound in the lower regions of the thorax; the influence of the similar locomotion of the stomach and intestines will vary with the filled or empty state of those viscera. The heart pushed downwards and inwards, and receding somewhat from the chest-wall during inspiration, where its place is taken by the inflated lung, gives dulness of less extent and lower site than in expiration.

In consequence of this triple influence of the respiratory movements on the results of percussion, it is advisable, under all circumstances, and absolutely necessary in delicate cases, that the act of respiration be at the same stage of progress, when the two sides of the chest are percussed comparatively. The end of a full inspiration is in such instances the fittest moment for striking; as, by desiring the patient to hold his breath, we may then be certain of having both lungs in the same state for a short while. If the respiration be calm, however, such nicety is not required.

In the state of health the posture of the patient (except in so far as it may interfere with the act of striking on the part of the physician, or alter the tension of the patient's own muscles, or the relative position of the subcutaneous tissues,) does not *directly* affect the results of percussion of the surface, wherever it corresponds to lung-substance. In other words, ordinary changes of posture have no notable influence in modifying the relationship of the lungs and their containing walls to each other, or in altering the amount of air they contain, or are capable of containing. But as variation of posture very sensibly alters the position of the heart, either in a downward, forward, or sideward direction, the posture of the patient must always be taken into consideration in estimating the results of pulmonary percussion near the cardiac region.

Variations from the Standard Type of Pulmonary Percussion compatible with a Healthy State of the Thoracic Organs.

The sound yielded by the chest of different individuals varies in clearness; being, generally speaking, clear in proportion to the thinness of the walls. In accordance with this it becomes distinctly clearer in persons who, from a previous state of fatness, fall into one of emaciation.

The sound varies, too, with age. From the age of four or five to fifteen (I have not sufficient experience among younger children, to make a positive assertion concerning them) the pitch is high, the duration marked, the resistance slight; the walls are flexible, elastic, and commonly not much loaded with soft textures. The ossification of the cartilages, desiccation of the ligaments, and general stiffness of the thorax in old age impairs the freedom of vibration, and deadens the sound; and they would probably do so much more generally than is actually observed, were it not for the frequency of atrophy of the parenchyma of the lung in advanced life: wasting of the external soft parts, too, tends to the same modifying result.

Marked deformity of the chest, congenital or acquired, impairs the resonance over lung, healthy in itself. Broken ribs, too, lessening freedom of motion on the affected side, and hence lessening the quantity of air in proportion to the pulmonary substance beneath, deadens the percussion-sound,—a fact showing that slight dulness does not, after an injury of the kind, absolutely prove the existence of pulmonary congestion.

Unusual laxity of the ligamentous structures of the thorax renders the walls unfavourable to vibration. But, making all allowance for the various conditions referred to, there will still remain instances in which the sound is unusually clear or dull, without its being possible to assign any satisfactory cause for the peculiarity. The real existence of such cases, however inexplicable at present, should always be borne in mind by the clinical observer.

Variations in the Results of Percussion, dependant on Disease.

Whatever be the nature of the morbid conditions of the sound discovered in any case, the space in which they exist may be either accurately defined or not; in other words, the morbid state, whatever it is, may either gradually pass into the healthy, or cease abruptly. In the latter case there is no difficulty experienced in ascertaining its precise limits; in the former, there often may be some indecision on the point, and it will be found useful to glide the pleximeter-finger *rapidly* over the entire region under examination, continuing the percussion all the time it is moved. In this way the exact line at which dulness or other change begins may be detected in very difficult cases.

§ I.—ALTERATIONS OF SOUND.

I.—STATICAL SIGNS.

The statical changes in the percussion-sound, produced by disease, are comparatively few in number, and simple in nature, but the indications they furnish most precise and valuable. Concisely stated, these changes are :—

1. *Diminution of clearness*, gradually passing into perfect dulness,—the *duration* of the sound being proportionally *shortened*, and the *sense of resistance increased.*

2. *Increase of clearness and of duration*, with *decrease of resistance.*

3. *Increase of clearness and of duration*, with *increase of resistance.*

4. *Alterations of quality.*

1. Diminution of clearness, with its attendant changes, occurs wherever the density of the materials, underneath the part struck, is increased. Thus, deficient resonance exists, *first*, wherever any new material, of greater density than lung and air combined in the natural relationship, has accumulated within the

chest: whether it be in the lung proper, as in congestion, inflammation, abscess, gangrene, serous infiltration, apoplexy, simple chronic consolidation, infiltration or tuberous accumulation of exudation-matter, tubercle or cancer within the parenchyma;—or in the pleura, as in cases of hydro-thorax, and hydro-pneumo-thorax, hæmo-thorax, pleurisy in the periods of exudation, effusion, and retraction, empyema, serous infiltration of old false-membrane, and solid adventitious products of all varieties;—or in the bronchi, as in cases of abundant muco-purulent accumulation within the tubes;—or in the mediastina, as in cases of hypertrophy of their cellulo-fatty tissue, enlarged bronchial glands, abscesses, and adventitious solid products. *Secondly*, such deficiency arises where any condition (material or dynamic) exists, favouring expiration and impeding inspiration: as in obstructive diseases of the upper air-passages, spasm of the glottis, and spasmodic asthma,—in the latter affection the presence of pulmonary emphysema often prevents the failure of resonance from being discovered. *Thirdly*, dilatation and thickening of the bronchi, condensing the adjacent parenchyma, produce a similar effect. It is obvious, too, that serous infiltration of the parietes, abscesses, tumors, thickening of the periosteum of the ribs in syphilitic persons (a state of great importance clinically) will deaden the sound. The influence of the heart and great vessels will be elsewhere considered.

The characters of the dulness are not *per se* capable of revealing its cause, at least with very rare exceptions, in the present state of knowledge. Still the very intensity of the dulness and great resistance go far sometimes towards establishing the diagnosis in cases of intra-thoracic tumor. Between this extreme and the opposite one of slight impairment of sound in spasmodic asthma, all shades of difference may be observed.

The superficial extent and locality of the dulness, important guides to the detection of its cause, will be dwelt upon in the description of individual diseases. It may be remarked here, however, that the area, within which loss of resonance is detected, may either be *fixed*, or *changeable with the position of the patient.* The former is infinitely the more common case; no matter how the posture be varied, the line of demarcation of the naturally and morbidly sounding parts commonly remains unaffected. But under certain comparatively rare circumstances, the boundaries of the dull sound may be more or less completely altered by causing the patient to vary his posture; the infra-scapular region, which may have sounded dull when he lay on his side, or reclined backwards, will give a clear sound, after he has remained leaning forward for a short while, and *vice versâ.* This moveableness of the sign indicates moveableness of its cause; and fluid collection in the pleura (from pleurisy, hydro-thorax, and perhaps hæmo-thorax) is the only physical state of which the percussion-signs have, hitherto, been found thus characterised; the fluid will, of course, gravitate to whatever part of the patient's chest his changes of position render the most dependent. It is, however, by no means so constant a sign of pleuritic effusion as might, from *à priori* considerations, be expected. The liquid is retained *in situ* in some cases by adhesions, which easily explains the fixedness of the dull sound; in other instances, the explanation cannot be found.

I have recently met with a case in which a massy encephaloid tumor hung by a sort of peduncle of condensed lung-substance to the surface of the organ, and so loosely that it must have altered its position as the patient turned from side to side.* But there was enormous pleuritic effusion present on the patient's admission, which would of course have prevented the discovery of any change of place of the tumor, had the idea of looking for such locomotion occurred to me: the source of fallacy must be infinitely rare.

2. Increased clearness and duration of sound, with decreased resistance (or excess of elasticity), is noted, where the relative quantity of air within the chest is increased, but not carried to such extremes as to interfere, by tension of the walls, with their

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* U. C. H. Dewing; Case Books, Males, vol. v. p. 19. Museum, No. 4000.

vibration,—in what may be familiarly called the *rarefying* classes of diseases, namely pneumo-thorax, hydro-pneumo-thorax (here in the upper parts of the chest), in atrophy, hypertrophy, and emphysema of the lung. It is possible that extreme anæmia, as suggested by Dr. Stokes, by lessening the relative quantity of blood in the lung, may increase the clearness of the percussion-sound.

In hydro-pneumo-thorax the air and fluid may sometimes be made to change relative positions, to a certain extent, by changing the patient's posture; the site of the exaggerated resonance will (unless adhesions interfere) always be towards the upper surface.

3. Increase of clearness and of duration of sound, with increased resistance of walls, is observed where there is a surplus of air in the subjacent part, with considerable induration of tissue between the surface and the part containing that surplus, —a combination of conditions met with where a superficial cavity in the lung has a thin, indurated, and adherent external wall.

4. As far as it can be rendered intelligible by words, the nature of the above three classes of alteration is explained by their names; the case is different with the fourth class, that of alterations of quality, which consequently require fuller description.

Instead of the quality *sui generis* which distinguishes the natural sound emitted by the chest, others, assimilable to those of various well-known tones, exist in certain states of disease. These morbid varieties of quality are referrible to three types: (*a*) Wooden; (*b*) Hollow; (*c*) Tympanitic.

(*a*) *The wooden* quality is very closely that of the sound yielded by mediate percussion of a common table, and distinctly conveys the idea of hardness. The duration of the sound having this quality is commonly less than in the natural state, and the sense of resistance experienced by the fingers is unusually great. I am inclined to think that, when well marked, it may be considered almost a conclusive sign of a thick and dense stratum of fibrous substance in the pleura, binding the lung and parietes together. No amount of fluid in the pleural cavity, or of consolidation of the lung itself, seems capable of producing it to a marked amount; but all descriptions of consolidation, when indurated false membrane is present in abundance, furnish it more or less.

(*b*) The *hollow* type occurs under three varieties,—the tubular, amphoric, and cracked-metal.

The *tubular* quality is precisely like that of the resonance emitted by the trachea under mediate percussion. The sound it characterises is of some duration, and the thoracic elasticity somewhat impaired. This character is to be detected (always more readily if the patient's mouth be kept open) about the inner part of the infra-clavicular region in cases of pleuritic effusion occupying the lower parts of the chest,—sometimes during the height of the disease,—sometimes, when absorption has just commenced; the larger bronchi are brought in this situation unusually near the surface, and so within the direct reach of the impulse of percussion, especially as they are surrounded by condensed lung. Again, it is observed in the stage of retraction of the same disease, when the plastic matter during its contraction may be supposed to have altered the relations of the lungs to the walls, and brought the tubular part of the former (the bronchi are often dilated too) into undue proximity to the latter. So, too, when solidified lung (acute or chronic), pent-up collections of pus, or solid products of any kind lie between the large bronchi and the costal surface, the sound acquires this quality. Mere dilatation of the bronchi will have the same effect. The internal topography of the chest explains why this sound should be most common in the infra-clavicular and upper mammary regions; but it may occur in the inter-scapular region from enlarged bronchial glands,* or over a great part of the back in cases of intra-thoracic tumor.† Excavations in the lung of small and medium size furnish similar resonance.

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* Petrolini: U. C. H. Case Books, Females, vol. v. p. 176. Here the tubular quality, distinct on gentle, disappeared on strong, percussion.

† Nature and Treatment of Cancer; case of S. C., p. 362.

The *amphoric* quality (the tubular on a larger scale) is heard in typical perfection in the sound produced by filliping the cheek, when the mouth is closed and fully, but not too tensely, inflated,—and also in the percussion-sound of the left infra-axillary region in certain states of distension of the stomach. I have observed this perverted quality over the entire upper front of the chest, including the clavicle and first bone of the sternum, in a few cases of pleuro-pneumonia, and in as great purity as it is ever heard over the stomach. When of this origin, the sound does not last long. But the common source of the sign is a cavity (tuberculous generally) of large size, near the surface, and provided with hard and thin walls.

The *cracked-metal* quality (*bruit de pot fêlé* of Laennec) somewhat resembles that of the sound resulting from striking the back of the hands, loosely folded across each other, against the knee,—the contained air being forced out quickly and abundantly between the fingers at each blow. When this character modifies the percussion-sound of the chest, there is coupled with it almost invariably a good deal of the amphoric quality, and the combination gives a result altogether *sui generis*, which, once heard, cannot easily be forgotten. Let there be a pulmonary cavity of large size with anfractuous walls, and communicating freely with the bronchi, the corresponding parietes being at the same time particularly yielding, and percussion will elicit this variety of quality. It is commonly said to depend on the collision of liquid and air produced by the blow ; but the sudden propulsion of air (forcibly expelled from the cavity) against the walls of the passages with which it comes in contact, seems sufficient to generate it. In the first place, cracked-metal resonance is producible over cavities, when free from fluid,—at least if the absence of cavernous rhonchus is to be trusted to as evidence. In the second place, if, as I found several years ago,* the nose and mouth be tightly closed in a patient furnishing the cracked-metal sound when they are open, that character will at once cease to be producible, though percussion continues to give an amphoric note. The fair interpretation of this fact seems to be, that the sudden rush of air from the cavity outwards produced by the forcible blow upon the yielding parietes in the ordinary open state of the mouth and nose, but completely prevented by their closure (the only condition materially altered in the experiment), is the real cause of the phenomenon. Whether the communication with the external air be interrupted or not, the contents of the cavity may be dashed together by percussion: were the common notion correct, the cracked-metal character ought therefore to be elicited in both cases. This explanation derives support from the conditions of production of the amphoric and cracked-metal sounds by striking the hands, closed so as to form a hollow, against the knee: if they be so closed as to prevent air from being forced from between them by the blow, the amphoric character only is detected,—if air be allowed to escape freely, the character analogous to the cracked metal is superadded; yet here, certainly, there is no *liquid* to take part in its production.

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* Lancette Française, 1834.

It has been well observed by Dr. Stokes, that a "metallic resonance, somewhat analogous to the cracked-jar sound of cavities, but evidently more diffused," is occasionally discoverable in cases of bronchitis, particularly in children. I have repeatedly observed this in infancy; it becomes especially likely to mislead, when there is evidence of tuberculous disease in extra-thoracic organs, as the brain or meninges.* The sound is certainly more diffused than, and otherwise different from, that dependent on excavation; but the absence of other cavernous phenomena, and the knowledge of the fact that bronchitis may simulate (for it is no more than this) the sound, are the best safeguards against error. The pliancy of the chest-walls explains the peculiarity in infancy. I have known a tubular note in pneumonia become distinctly cracked-metal when consolidation was at its height.—(Pneumonia of right apex, U. C. H., Feb., 1849.)

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* Case of Eliza Wright, æt. 4; U. C. H. vol. v. October, 1849.

(c) The *tympanitic* character, as its name signifies, resembles that of the sounds of a drum. The note is, generally speaking, very clear; the duration considerable; the resistance of the walls tense, drum-like, highly elastic. Totally different in character and mechanism from the varieties of the hollow type of the percussion-sound, it has, nevertheless, been confounded by numerous foreign writers with the amphoric variety: it is pulmonary quality exaggerated.

It exists to the maximum degree in pneumo-thorax and hydro-pneumo-thorax, with considerable distension of the side; if, however, the distension be extreme, so as to interfere with chest-vibration, and produce actual compression of the contained air, the amount of tympanitic quality lessens.* In emphysema, with bulging of the chest, it is also occasionally observed, but never in the perfection attained in pneumo-thorax. Further, as was first noticed by that accomplished observer, Dr. Graves, the quality of the note over pneumonic consolidation, sometimes, temporarily becomes tympanitic: of this I have now observed two positive examples,† in one with, in the other without, plastic lymph on the pleural surface. The only plausible explanation seems to be, that air, the product of secretion, accumulates for a time (in my cases for a few days) in the pleural cavity.

II.—Dynamic Signs.

It has been seen, that in health the act of respiration modifies the results of percussion in three different ways; now in disease the natural modifications may be perverted or impeded, and hence a certain number of dynamic signs. Few of these signs are of practical importance; but occasionally some of them prove valuable.

1. As regards the increased volume of the lung, and the consequent extension of pulmonary resonance attending inspiration,

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* *e. g.* Plympton; U. C. H., Males, vol. iv. p. 410.

† Adventitious Products, Cyclopædia of Anat., p. 145.

neither will occur in cases of hydro-thorax, pleuritic effusion, and pneumo-thorax, in emphysema, and obstruction of the chief bronchus, either from foreign bodies or inspissated mucus within it, or from pressure, aneurismal, glandular, or other, without it. In a remarkable case of the latter kind, where the respiration was permanently weak, the failure of inspiration in increasing or extending the resonance of the side, contributed much to the diagnosis of obstruction of the main bronchus.* In such cases, too, as during expiration the air, which in health would escape, still stagnates within the chest, there will not be a sufficient reduction of the area over which pulmonary resonance is discoverable.

2. *Mutatis mutandis*, the respiratory influence on the clearness of percussion may be similarly affected. In health, full inspiration increases the clearness of the sound of percussion, and equally so on both sides of the chest. Certain states of disease, impeding full pulmonary expansion on either side, interfere on that side with the production of the increased clearness discoverable on the other after full inspiration;—hence a sign founded on *comparatively deficient increase of clearness at the close of a full inspiration* on either side, the sound being equally clear on both in the ordinary state of respiration. Again, the sound of the healthy chest is somewhat deadened by full expiration, and equally so on both sides of the chest. Certain states of the lung remove this equality by rendering the sound disproportionately dull in the situation where they exist: hence the sign of *comparatively great diminution of clearness at the close of full expiration.* Both these delicate signs sometimes give very satisfactory results in cases of small, irregularly scattered indurations, tuberculous or other, of either apex. The mechanism of the former is obvious; the latter depends on reduction of size of the lung, in expiration, bringing within a small space solid matter which had previously been more widely scattered.

Or, on the other hand, other states of the lung, by impeding the expulsion of air from the vesicles, render the sound disproportionately clear; hence the sign of *comparatively deficient diminution of clearness at the close of full expiration*, a sign discoverable in emphysema and obstructed bronchus. If there be air in the pleura, the same result will occur.

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* Case of Mary Ransom; U. C. H., Nov. 1848.

3. The cases in which respiration ceases through disease to exercise any influence on the position of the heart and abdominal viscera may be inferred from the foregoing exposition.

§ II.—ALTERATIONS OF RESISTANCE.

In his introductory observations upon physical diagnosis in general, Laennec makes a passing allusion to the "sense of elasticity perceived" in percussing; but in no instance refers to the diagnostic indications derivable from changes in this elasticity. Piorry and others have availed themselves of them fully, but their importance is not generally appreciated. There are cases of not very rare occurrence, in which erroneous inferences would almost inevitably be drawn from the sound elicited by percussion, were these not corrected by the information derived from the degree of resistance felt by the fingers. Take the case of a cavity seated close to the surface: the unnatural clearness of sound which sometimes distinctly exists over such cavities (quite independently of tubular or amphoric quality) might not only lead to an incorrect estimate of the state of the subjacent part, but also to the inference that the lung, in reality least affected, was the most diseased. The sensation of hardness and firm resistance experienced by the fingers at once discloses the true cause of the unusual clearness. Besides, the cases are extremely numerous in which it is satisfactory to have the corroborative evidence, furnished by the state of resistance, in favour of the inference drawn from the sound. That doubt often exists as to the state of the sound on the two sides, is unquestionable; and in these cases the condition of the subjacent parts may frequently be settled by taking into consideration the amount of resistance. To those persons whose sense of touch is more delicate than that of hearing, this source of diagnosis is of especial value.

In describing changes of sound, a good deal has been incidentally said on those of resistance; but recapitulation is advisable. The resistance depends on the state of the contents and of the walls of the chest. All conditions lessening the relative quantity of air in the lung, while they deaden the sound, increase the resistance; they have already been enumerated. All conditions which conversely increase the relative quantity of air, decrease that resistance, while they increase the clearness of the sound; these also have been enumerated: pneumo-thorax is the typical disease. But the walls of the chest may be so stretched in that very affection by excessive accumulation of air, that the resistance becomes extreme, though the sound continues clear. Here the state of the walls themselves modifies the resistance; as it does likewise where solid or liquid matters accumulate in their substance, where the periosteum thickens, or the ribs either enlarge, or (as in the retraction-period of chronic pleurisy) become unduly approximated to each other.

SECTION V.—AUSCULTATION.

AUSCULTATION means the act of listening, and is termed pulmonary, cardiac, &c., according as the sounds listened to are produced in the lungs, or in the heart, &c.

The direct object of pulmonary auscultation is the appreciation of certain sounds audible on the external surface of the chest, and either produced by the respiratory play of the lungs themselves, or transmitted in a modified form by these organs from others, as for example the heart, in which they are actually evolved.

The method of performing auscultation may be *immediate* or *mediate:* in the first case the ear is applied directly to the chest; in the second, an instrument of variable material and construction (originally a hollow cylinder of wood, to which the name of stethoscope* was given by Laennec, and has been retained for its modifications,) is used as a conducting medium between the surface examined and the ear.

Both of these methods of auscultation have had their favourers and their detractors. The advocates of mediate auscultation urge that—The stethoscope can be closely applied to several points of the chest (axillary, upper scapular, inter-scapular regions, and acromial angle, &c.), where the ear cannot be placed in accurate contact with the surface; †—the use of the stethoscope enables the observer to auscult in a posture more easy to himself (a point of much importance for securing correct results) than that he is obliged to assume if he apply the ear directly;—it is indelicate to place the head upon the persons of females;—it is disagreeable to bring the head in contact with the bodies and clothes of some of the lower orders;—the limits within which the various sounds are perceived are more nicely circumscribed with the stethoscope than the unassisted ear;—certain phenomena, as pectoriloquy, are more distinct when the stethoscope is employed. On the other hand, while the partisans of immediate auscultation admit that in lean persons it is difficult to place the ear appropriately in some few situations, they affirm that such cases very rarely occur, and that in all others the stethoscope is an *inutile lignum,*—the direct application of the ear giving as precise indications as the employment of that instrument, with less appearance of fuss and less real trouble. For my own part, I entertain no doubt that Laennec and others have greatly exaggerated the superiority of mediate over immediate auscultation in respect of the distinctness with which the phenomena are heard, and the precision with which they are circumscribed, in cases where *both* modes of auscultation *can* be employed; and that this distinctness and this precision are in fact greater in such cases with mediate or with immediate auscultation, according as the observer is more habituated to one or other of these modes of examination. It seems very plain, however, that as there are cases in which the ear cannot be directly applied, or in which it is disagreeable or indelicate to do so, mediate auscultation is the method with which the student should most closely familiarise himself; while, on the other hand, as it is often difficult to persuade children to allow the stethoscope to be applied, and as we may often desire to auscult an adult when no instrument is within reach, the ear should be practised in immediate auscultation also.*

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* From *στηθος* 'the chest,' and *σκοπειν* 'to examine.'

† These points are still more numerous in malformed chests.

The proper construction of the stethoscope has been a subject of constant dispute. As was felt by Laennec, theory, in the present state of acoustics, deposes in favour of the solid instrument; yet, as a hollow one is almost universally employed, it may be inferred that theory is somewhere unsound. From trials with hollow and solid ebony and cedar stethoscopes, I have come to the following conclusions:—That with the hollow instrument the respiratory murmurs appear stronger and more open in quality,—with the solid, weaker and sharper, so much so that a bronchial character may be simulated; that cavernous phenomena lose in some measure their hollow quality with the solid instrument; that friction phenomena are sometimes materially better heard with this; that the natural vocal resonance over the trachea is hollower, graver, and better articulated with the hollow cylinder; and that the resonance of the observer's own voice, as he speaks, while ausculting the chest of another person (autophonia), is materially more intense with the solid than the hollow instrument.

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* Beyond all doubt, first-rate skill in *pulmonary* auscultation might be acquired without using the stethoscope at all, though it is common to hear the *invention of the stethoscope* spoken of as constituting Laennec's claim to immortality. No! his name will be imperishable, because he *discovered auscultation*, (for the ideas of Hippocrates, Hook, and Double may, without injustice, be ignored,) described accurately the sounds it detects, and traced these sounds to their anatomical conditions. Persons, indeed, are to be found, who seem to think that the stethoscope possesses some mystic faculty of communicating diagnosis, and saving the auditor all trouble of thought; unfortunately, the instrument is no more than

> . . . die todte Sprachrohr, das den Schall
> Empfängt und wiedergiebt, und selbst nicht höret.

Glass, gutta-percha, and metal have been tried, but are certainly inferior, for various reasons, to wood (cedar or ebony). The really important point is, that the ear-piece should fit the ear well,—it is as necessary to try on a new stethoscope, as to try on a new hat. Beyond this, all depends on the use to which the student puts the instrument, and not on the density of the wood, the direction of its fibres, &c.

In performing auscultation, several precautions, affecting the observer and the observed, are to be attended to. 1. The chest should be uncovered; or if, from circumstances, such exposure be inadmissible, as thin a layer of clothes as possible allowed to remain between its surface and the stethoscope. 2. All friction between the stethoscope and the patient's or the observer's clothes should be carefully prevented. 3. The position of the patient should be regulated in the same manner as for the performance of inspection; an unconstrained state of the muscles being particularly necessary, in order to insure free entry of air into the lungs. The sitting posture is, every thing considered, the most conducive to perfect investigation, provided the chair employed have a tolerably high seat, and the observer be of the middle height; a tall person will find himself most at his ease if the patient stands. It of course frequently happens that the recumbent posture is the only one the patient can easily assume, —he may be perfectly unable even to turn on either side. Under these circumstances the utility of the flexible stethoscope has been insisted on, as it may be applied far back laterally, and even to the dorsal regions, if the body be inclined a little sidewards, without moving the patient. I confess I have never yet seen an ordinary case of pulmonary disease, whether primary or secondary, (of the latter, the pneumonia of continued fever is the example most to the point,) in which, so long as it was a matter of importance to auscult the chest, the patient might not be raised sufficiently by careful attendants to admit of the examination being efficiently made, for practical purposes, with the common stethoscope or ear alone. But attendants may not be within reach; and, in special cases, tendency to syncope may make it dangerous to raise the patient's head;—here the flexible instrument will be useful. If in the sitting posture, while the front of the chest is submitted to examination, the patient should sit not exactly erect, but with the trunk sloping a little backwards, the arms being allowed to hang loosely at the sides. When the observer proceeds to examine the lateral regions, the patient may be directed to clasp his hands on the top of the head,—in other respects, retaining his former posture; and, lastly, when the dorsal regions are examined, sit upon the chair sidewise, (if a male, astraddle,) with his back to the observer, his arms crossed, and his head bent somewhat forwards. *Mutatis mutandis*, the same precautions are to be taken when the patient stands, lies, or sits up in bed. 4. It is of importance to apply the stethoscope firmly, but not forcibly, to the surface: too slight or too strong pressure interferes with the accurate transmission, or alters the character, of the sounds. Thus, it is well known that Laennec exaggerated the frequency of ægophony; and it has been plausibly conjectured by M. Fournet that his error arose from the habit he had contracted, and which he recommended to his pupils, of applying the ear very lightly to the stethoscope, when searching for that modification of resonance. At least it is certain that an ægophonic character is sometimes given to a natural resonance by adopting this plan. On the other hand, persons with tender skins, or in a state of emaciation, cannot endure rough application of the instrument. 5. Great care must be taken to insure accuracy of contact between the skin and every point of the circumference of the end of the stethoscope; as a necessary condition for this, the instrument must be applied perpendicularly to the surface, and held until firmly placed, by its applied end: the auscultator may then readily assure himself with the fingers, whether the skin and the edge of the instrument are in accurate apposition. 6. The posture of the observer should be free from all constraint; he should apply his ear to the stethoscope with as much care, as the instrument to the chest; concentrate his attention upon the sound examined; and, unless he be thoroughly experienced, proceed (as far as is compatible with the patient's safety) slowly with his examination. The motto *festina lente* is a good one for the beginner in the study of physical diagnosis. 7. It is advisable to commence the auscultation of patients, while they breathe in the manner to which they are naturally inclined; because it is important to ascertain the precise natural condition of the respiration, and besides, directions for the regulation of the act often puzzle. Some individuals, however, absolutely require guidance; as the moment they perceive the instrument applied to their chest, they throw the muscles of the trunk into violent and unnatural motions, which of course materially impede the entry of air into the lungs. The simplest way of making such persons breathe in an efficient manner is, to perform several quick *noiseless* respirations before them, and desire them to imitate these. This method will, however, occasionally fail; our object may then be gained by desiring them to sigh, to speak, or to cough. The deep inspiration required for the performance of these acts will at once enable the observer to ascertain the condition of the murmurs; and, indeed, there are many states of the lung in which, quite irrespectively of the patient's manner of breathing, much information may be gained by a single cough. 8. The sounds produced in the pharynx by the passage of the air are liable to be confounded with the true pulmonary sounds of respiration; the error may be avoided by directing the patient to open the mouth, if it have been previously shut, and *vice versâ*. If the sounds heard have their seat in the lungs, they will suffer no notable change from this opening or closing of the mouth; if in the pharynx, they will be more or less modified in character. It is, however, only in unusual cases that a direction of this kind is *necessary* to enable a practised auscultator to avoid the error referred to; though it may often be advisable to corroborate thus the impression derived from ordinary examination. The sensation of *distant* production which attends the pharyngeal murmurs, and the occurrence of a distinct interval of time between inspiration and expiration (a point to which I particularly recommend attention), will suffice to distinguish them from the true pulmonary sounds. With ordinary watchfulness, the observer may distinguish the two kinds of sound at the same time. 9. Both sides of the chest must be submitted to precisely the same examination,—conducted precisely in the same way,—as already explained in reference to percussion. 10. Auscultation should never be considered complete, until the entire chest has been examined; it is often in some other situation, where the symptoms would least have taught us to look for disease, that auscultation proves its existence. 11. In acute affections, auscultation should be repeated twice, at least, in twenty-four hours. 12. The student should accustom himself to the use of both ears.

PULMONARY AUSCULTATION IN HEALTH.

The sounds discoverable by auscultation of the breathing-apparatus in the state of health are:—(A.) The respiratory murmurs; and (B.) The resonance of the voice.

(A.) *The natural respiratory murmurs.*—Two sounds, audible by immediate or mediate auscultation, attend each act of healthy respiration; one, corresponding to the movement of inspiration, the other, to that of expiration. These are the *inspiratory* and *expiratory murmurs* or *sounds.*

The *essential or primary properties* of these murmurs, practically considered,—those which, in their modified states especially, possess diagnostic importance,—are their: 1. *Special Character and Quality*; 2. *Pitch*; 3. *Intensity*; 4. *Duration*; 5. *Liquidness*; 6. *Softness*; 7. *Rhythm.*

By the *special character* of a sound is understood that essential peculiarity which must, under all circumstances of intensity, duration, rhythm, &c., distinguish it from others; the special character of the sounds of a piano-forte, for example, will invariably differ from that of the tones of a violin. Here also, for the purpose of simplifying the object as far as possible, without incurring any material sacrifice of accuracy, may be included that property of sound known as *quality*; though, in point of fact, the *quality* or "timbre" of a sound is a different thing, acoustically speaking, from its *character*. Thus, two *tenor* voices (identical therefore in point of character, and accurately so, also, of register) may sound the *same note*, in the same *rhythm*, with the same amount of *liquidness*, with the same *intensity*, and for the same *duration* of time, and yet a marked difference shall exist in the sensations impressed upon the ear by the two tones. Difference in quality is supposed to depend on the form of the sonorous waves; but the conditions determining that form have not been ascertained. As the quality of the notes of musical instruments varies with the properties of the material composing them (the secret of the Cremona violin is in the wood), it may reasonably be conjectured that the tension, dryness, elasticity, and other properties of the tissues engaged in the production of laryngeal and pulmonary sounds modify the quality of these.* The *pitch* of sounds rises as the frequency of the vibrations in a given time of the sonorous body; the evident variations in pitch of the respiratory murmurs under different circumstances immediately depend on variations in that frequency,—but why, or through what mechanism, the frequency is affected by different anatomical conditions, is unknown.

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* Some of the conditions regulating the quality of the human voice are theoretically under the influence of the will; and, as matter of experience, it is known that effort—and imitative effort especially—will modify the quality of singing tones. Thus, I know a tenor singer who manages during the opera-season to throw into his voice (one of ordinary tenor character) a something of the marvellous quality of Mario. This person assures me, that the power (which disappears with the disappearance of his model) comes of imitative effort.

The terms *intensity* and *duration* explain themselves. The notions of *dryness* and *liquidness* of sound may be at once obtained by squeezing close to the ear first a perfectly dry, and then a moistened sponge. Similarly, if we press together a mass of wool held beside the ear, the property of *softness* in sound will at once become intelligible; its converse, *hardness*, by grating together any two hard bodies. The *rhythm* of a sound means its mode of progression or evolution, which may be continuous and equable, or interrupted and jerking.

Although in originally establishing the varieties of morbid respiration, every respiration-sound requires to be analysed in respect of these various properties, the complexity of the matter is much less in actual practice than it seems; for experience proves that several of these properties are almost invariably altered simultaneously, and of course such *compound states* may be described for convenience sake by single phrases.

The properties of the murmurs differ in the various divisions of the respiratory organs; for each of these divisions there is a healthy type of respiration, commonly termed, a. *pulmonary* or *vesicular;* b. *bronchial;* c. *tracheal;* d. *laryngeal;* e. *pharyngeal;* according to the part of the respiratory passages from which the sounds audible externally are transmitted.

The sole point in which these varieties of respiration agree is, that in all of them the audible sound may be resolved into two—an inspiratory and an expiratory. From their numerous distinctions they require separate consideration.

a. *Pulmonary.*—The pulmonary *inspiratory* murmur is a sound of gentle breezy character; neither liquid nor dry; soft; of a certain intensity and duration; and in respect of rhythm, gradually developed and continuous. The *expiratory* murmur, slightly harsher and hollower than its predecessor, and of lower pitch, is about three or four times the weaker and shorter of the two, and in some persons (especially at the left side) is actually inaudible.*

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* The fact that the escape of air from the lungs during expiration is com-

The term "breezy" seems the fittest by which to describe the *character* of the healthy respiratory murmurs. They suggest in their pure state the sighing sound of the breeze among leaves, the only difference being one of intensity. The use of the term *vesicular*, in speaking of the natural condition of these sounds, has led to an erroneous impression. It was originally applied to designate the *seat* of their production; but not a few persons have incorrectly referred the term to the *character* of the sounds. There is nothing in the nature of the respiratory murmurs suggestive of a connection with vesicles; and, whenever such character occurs, the phenomenon it attends is morbid.

The two murmurs so closely follow each other in each healthy respiration, that they may, practically speaking, be said to be continuous. This continuousness, indeed, forms an important character of *pulmonary* respiration of *healthy* type. It would of itself be sufficient to announce the lung as the part ausculted; for it will be found that in proportion as auscultation is practised, in health, at a further point from the pulmonary parenchyma, so will the two sounds be more and more distinctly separated from each other by an appreciable interval of time,—an interval which consequently attains the maximum opposite the larynx and upper part of the throat.

monly attended with audible sound was known to, and is distinctly stated by, Laennec. But the importance of the expiratory murmur, the valuable indications its modifications afford in the diagnosis of disease, did not sufficiently attract his attention; and to the late Dr. Jackson, jun., of Boston, U.S., belongs the credit of conceiving the value and extent of information which might be obtained from its analysis. In a most ingenious paper, read in 1832 before the Medical Society of Observation of Paris, that zealous inquirer forcibly drew attention to the subject. M. Louis and several of his pupils submitted the remarks of Jackson to the test of observation: his announcements were found generally correct, and thenceforth the separate consideration of the expiratory sound became with them habitual. Dr. Cowan subsequently favoured the English public with a valuable paper on the subject (*Lond. Med. Gaz.* vol. xviii. p. 332); and M. Fournet has since investigated it more thoroughly than his predecessors, and has popularised what had previously been known only to the comparatively few: but he has fallen into repeated errors of over-refinement.

What is the site of production of the pulmonary sounds?—and what is the mechanism by which they are generated? M. Beau* endeavoured to show that the respiratory sounds, heard on the surface of the chest, are not produced in the lungs; but are simply the pharyngeal sounds, attending the entrance and exit of air through the pharynx, transmitted through those organs. Two facts seem to disprove this theory;—the existence of an appreciable pause between the murmurs opposite the pharynx, while there is none such over the pulmonary structure; and the occasional possibility of hearing at one and the same spot of the chest-surface, ordinary respiratory sounds and pharyngeal sounds. Besides, the two pharyngeal sounds are equal in length, the pulmonary extremely unequal; and destruction of portions of the fauces does not alter or impair the pulmonary murmurs. The three first of these objections are equally fatal to Dr. Spittal's theory,† that in the glottis is the main origin of the sounds. The loud pulmonary murmur with which air makes its way into the lungs, through an artificial opening *in the trachea*, is well known; while, *per contra*, laryngeal and tracheal stridulous respiration is commonly attended with very weak pulmonary murmurs. Laennec believed that the sounds were caused by "the entrance of the air into, and its expulsion from, the air-cells of the lungs;" and this appears to be their essential cause. The terminal portions of the bronchi are also probably concerned in the sounds,—the vibrations of the moving air, and moving parenchyma, and the distension of the air-cells, all, doubtless, contributing to the general result. The excess of inspiratory over expiratory sound seems explicable by the greater resistance of the textures during inspiration, and by the current being in the former instance directed towards the ear. If a slightly moist sponge be placed between two stethoscopes, and one person breathe naturally into one of them, while a second auscults by the other, it will be found that the difference in force of the sonorous currents towards and from the ear of the auscultor is about the same as between the intensities of natural inspiration and expiration. And, again, if a sponge in the same state be held close to the ear, and alternately pressed and allowed to expand, it will be found that the closure of the cells of the sponge is almost noiseless, their expansion accompanied with well-marked sound: the ratio is very much that of the expiratory and inspiratory sounds.

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* Arch. Gén. de Médecine, 1834.

† Edin. Med. and Surg. Journal, vol. xli.

The conditions of the pulmonary murmurs may vary within certain limits from those just described, without the type of respiration ceasing to be compatible with health of the lungs. In other words, there are *healthy varieties* of respiration. They are referrible to the following circumstances: *age; sex; the part of the chest furnishing the sounds; the rapidity and fulness of respiration; temperament and idiosyncrasy.*

Age.—The description given of healthy respiration refers to that of adults; at either extreme of life its characters are different. In infancy the *intensity* of the sounds is considerably greater than at a more advanced age, all the other properties of both sounds remaining unaltered both positively and relatively (*puerile* respiration); the expiration-sound appears disproportionately long only from its greater intensity. The smallness of the vesicles at this age, and hence the more extensive surface, concerned in the production of the murmurs, seems to account best for the excess of sound in infancy. The slightly greater frequency of respiration in childhood seems inadequate to its explanation, especially as there is no such difference in the laryngeal respiration of childhood and adult age. In old age, on the other hand, the *intensity* of the sounds is much diminished, and the duration of inspiration lessened, while that of expiration is increased (*senile* respiration). Healthy *senile* respiration (as it may be called) differs from morbidly *weak* respiration in the increased duration of expiration. Andral has referred to its characters, and justly connects it with the rarefied and inelastic state of the lung which arises, as a condition of natural decay, in advanced life.

Sex.—The pulmonary murmurs are generally louder in the female than the male; and certain peculiarities, to be presently mentioned, are of more frequent occurrence in the former than the latter.

Region and Side of the Chest.—Laennec's opinion, that the murmurs are loudest, where the lungs approach during expansion nearest the thoracic parietes, is not very intelligible. The sounds are fuller superiorly than inferiorly (especially in women), and in front than behind. They are audible even at the bases of the right and left infra-mammary regions on full breathing. Between the scapulæ, and over the upper part of the sternum, the respiration presents more or less distinctly a *bronchial* character.

The characters of the inspiratory murmur do not differ in the corresponding points of the two sides of the chest to any appreciable amount: and there is no excess, as a constant condition, in the duration and intensity of the expiratory murmur in any one part of either side as compared with its fellow spot; but in a certain proportion of persons (especially females, as originally shown by M. Louis) the expiratory murmur is longer and louder in the supra and infra-clavicular, the supra-spinata, and the inter-scapular regions on the right side than the left. The nearer the inner part of these regions the comparison is made, the more marked the difference: while very notable near the sternum, it may be imperceptible at the acromial angles. The excess of expiration signifies an approach to bronchial character.

Rapidity and fulness of Respiration.—The *intensity* of the respiratory murmurs increases directly as the rapidity of respiration; their *duration*, as its fulness. When the respiration is at once full and rapid, both properties of the murmurs are affected simultaneously.

Temperament.—The respiratory murmurs are, generally speaking, of greater intensity in persons of nervous temperament, or labouring under certain nervous affections, as hysteria, than in persons otherwise constituted.

Idiosyncrasy.—In some individuals the respiratory murmurs are unusually weak or unusually strong, without its being possible to trace the peculiarity to any particular cause; it is then spoken of as an idiosyncrasy.

The proportional duration and intensity of the expiratory sound vary much in different chests; in not a few persons expiration is completely unattended with audible sound. This absence of expiratory murmur is, according to my experience, most frequent in males; and when it exists, is a natural peculiarity, and in no instance the effect of disease. The exception, which occurs to this statement in some cases of emphysema, is only an apparent one.

The student must remember that the muscular actions of the thoracic parietes themselves give rise to a peculiar buzzing, rumbling sound, especially audible if the patient require to use any effort to keep himself in the position he is placed in. The sound disappears if the muscles are relaxed; otherwise it is permanent. This is the only normal sound that can be confounded with the pulmonary murmurs.

b. *Bronchial respiration*, audible between the scapulæ, and at the upper part of the sternum, in the sites corresponding to the bifurcation of the trachea, wants in both murmurs the perfect softness and gentle breeziness belonging to the pulmonary species; both are slightly harsher (less so, however, than in morbid bronchial respiration), of higher pitch, more rapidly evolved, especially the expiratory, and follow each other less closely, are less accurately continuous, than in pulmonary respiration. In some persons the respiration is very purely pulmonary even between the scapulæ.

c. d. e. *Tracheal, laryngeal, pharyngeal respiration.*—The respiratory sounds, as heard over the trachea, larynx, and pharynx, are considerably more intense, less soft, drier, hollow in quality, somewhat whiffing, of higher pitch, and more rapidly evolved, though of greater duration, than in the divisions hitherto considered. While in pulmonary respiration the two sounds differ very materially in duration and intensity, in these upper sections of the system they tend to equalisation in these respects (the second perhaps slightly exceeds the first); and, instead of being almost continuous, are separated by an interval of some duration.

(B.) *The Resonance of the Voice.*—The particular rules for performing auscultation of the voice are not very numerous. It is of great importance that its intensity and tone be the same, while both sides of the chest are examined; and, in order to insure this uniformity, the observer may cause the patient to count a certain number of figures at a measured and even rate. Generally speaking, a loud tone should at the same time be directed; but in the auscultation of the voice over caverns, the most decisive information is sometimes obtained from the whisper, beyond which the patient is, in the advanced stages of tuberculous disease, frequently unable to raise his tone.

The stethoscope should be laid firmly upon the surface, and the ear similarly applied to the instrument, but without any degree of forcible pressure; if either be too lightly applied, a tremulous bleating character may be given to the resonance; if too forcibly, the distinctness of this is diminished. It is scarcely necessary to add, that the instrument should be used in precisely the same manner and in precisely corresponding spots upon both sides of the thorax. The condition known as exaggerated resonance is perhaps more accurately appreciable by means of immediate than mediate auscultation; all other unnatural states of vocal resonance are more satisfactorily and distinctly ascertained with the stethoscope. In examining certain regions, great care must be used both in the manner of applying the instrument and in drawing inferences from the results obtained; the chief of these regions are the post-clavicular, the upper sternal, and the interscapular. The vicinity of the trachea or large bronchi is, without such care, liable to mislead the observer, by the naturally greater resonance to which that vicinity gives rise. When the post-clavicular space is examined, especially its inner part, the stethoscope should be held as nearly as possible parallel to the trachea.

If the stethoscope be applied over the larynx or trachea of a healthy individual while speaking, the voice is transmitted, imperfectly articulated, through the instrument to the ear, with a degree of force, loudness, and concentration so great, that the experiment may be productive of actual pain to the observer, especially if the voice examined be grave and strong. The same transmission of the voice occurs at the lateral parts of the neck (and even over the spinous processes of the vertebræ behind), but with less intensity than on the middle line in front. The resonance of the voice heard in these situations is called *natural laryngophony* and *tracheophony*. At the upper part of the sternum, on the middle line, a considerable decrease of intensity in the natural vocal resonance, as compared with that just described, is observed; towards the edges of the same part of that bone a still further diminution is perceptible, and the resonance is here reduced to that state called *natural bronchophony*. The sound is now more diffused, while the voice appears to be (as it is) produced at a greater distance, and no longer *seems* to be directly transmitted through the instrument. This species of resonance is also observable posteriorly on the middle line over the division of the trachea, and on either side of that division between the spines of the scapulæ. Over the parietes of the chest, with the exception of the parts just referred to, the natural resonance of the voice amounts to no more than an obscure buzzing, unattended with the least approach to articulation, and appearing to originate at a certain depth within the chest, and over a certain extent of surface: in many persons even this obscure buzzing is wholly deficient.

The intensity and quality of this natural resonance are modified by certain circumstances altogether independent of disease. Thus, the natural resonance is *cæteris paribus* marked in proportion to the graveness of the voice. This statement is only true of intensity, however; there is no greater tendency to concentration or articulation of the sound when the voice is grave than when it is shrill: Secondly, the natural resonance of the voice is, as a corollary from the last proposition, more marked in males and in adults than in females and in children; it is also more marked in aged persons than in adults, probably on account of the thickening and hardening of the bronchi in old age: Thirdly, the quality of the resonance varies with the quality of the speaking voice; thus in people of advanced age it is very commonly tremulous and bleating: Fourthly, the natural resonance is more strongly developed, the larger the chest, and the less loaded its walls with fat and muscle: Fifthly, it is stronger in front than behind (with the exception of the interscapular region), and at the upper than the lower parts of the thorax: Sixthly, it is equal on both sides of the chest, except under the clavicles, and in the spaces between the spines of the scapulæ and median line; in these regions the phenomenon is more strongly marked on the right side, as first established by Sir James Clark and M. Louis, —Dr. Stokes incidentally mentions his belief (*Op. cit.* p. 497) that the same superiority extends to the *entire* right side: Seventhly, the intensity of vocal resonance, as of the respiratory murmurs, varies much in persons apparently presenting the same physical conditions for its development;—hence arises the difficulty, and commonly the impossibility, of drawing any inference directly from the state of vocal resonance in a given portion of the chest; it is only by the application of the principle of comparison of the two sides that any safe result can be obtained: Eighthly, if the voice be grave, the vocal resonance and fremitus are generally directly each as the other; if shrill, resonance may exist without fremitus.

PULMONARY AUSCULTATION IN DISEASE.

The sounds discoverable by auscultation of the lungs in disease are: — A. 1. Modified respiratory murmurs; 2. Adventitious sounds produced by the act of breathing; B. Modified vocal resonance; C. Resonance of the cough; D. Phenomena common to the respiratory murmurs, to rhonchi, to vocal and cough-resonance; E. The sounds of the heart and vessels, as transmitted through the lungs.

A. 1. *Modified Pulmonary Murmurs.*—It is extremely rare to find one only of the primary properties of the respiratory sounds affected; in by far the greater number of cases two or more of them suffer alteration at the same time; and thus are produced compound conditions of change, which may be described as distinct species of morbid respiration and classified as follows:—

Species of unhealthy respiration distinguished by changes of—

(α) *Duration* and *Intensity*; (a) exaggerated, (b) weak, and (c) suppressed respiration.

(β) *Rhythm* (either solely or in conjunction with other properties; (d) jerking; (e) divided; (f) altered ratio of inspiration to expiration.

(γ) *Quality* (and in addition other properties); (g) harsh and bronchial respiration, and (h) blowing respiration, with its varieties, simple, cavernous, and amphoric.

Each of these species requires separate consideration.

(α) *Changes of Duration and Intensity.*—(a) *Exaggerated respiration* is essentially distinguished by an increase in the intensity and duration of both murmurs, *especially the expiratory,*—an increase unattended with modification of any kind, either in respect of special character, softness, or liquidness. It is, likewise, termed *supplementary*, because the pulmonary tissue in which it is produced often supplies by increased energy the loss sustained by the inactivity of some other part; and *puerile*, on account of its similarity to the natural respiration of children. However, there is this difference between the supplementary respiration of the adult and the natural respiration of infancy, that in the former the expiratory sound is proportionally more affected. Undue prolongation of the expiratory murmur depending directly on disease, may be distinguished from that of exaggerated respiration, by its being attended with modifications affecting the *quality, softness,* and *liquidness* of the murmur.

The causes of exaggerated respiration are unnaturally rapid circulation of air through the lung, together with an increase in its quantity and the force of its impulse against the walls of the vesicles, and probably, in the *number of these expanded by each inspiration* in the spot;—all this being the result (except in very rare cases) of the inaction of some part of the same or of the other lung. Of very variable seat and extent, it occurs: (1). In healthy tissue, adjoining parts *obstructed* by bronchitis, new productions or foreign bodies in the bronchi; *condensed* by plastic effusion, pneumonia, tuberculous, cancerous, or simple consolidation, apoplectic effusion, &c.; or, *rarefied* by vesicular emphysema (2). In healthy tissue suddenly *released from spasm,* as in spasmodic asthma: (3). In tissue itself affected with *hypertrophy.* Exaggerated respiration, then, though not a *direct* result or sign of disease (except in the last rather doubtful case), furnishes valuable *indirect* evidence of its existence, and bears the same relation to the cause producing it, as (to use the happy expression of Fournet) the shadow to the substance. The closer the examination is made to the actually diseased part, the more intensely will the exaggerated respiration be found to be developed.

(b) *Weak respiration* is characterised by a simple diminution in the *intensity* and *duration* of the respiratory sounds, without change of their other properties. When the quality of the expiration is simultaneously altered, the respiration belongs to another type. There are two varieties of weak respiration; the *superficial* and the *deep-seated.* In the former the weak murmur appears to be produced on the surface of the lung or immediately under the walls of the chest; in the latter, at a greater or less distance from these walls. The physical conditions present explain these peculiarities: in the latter, the portion of lung in which the weak murmurs exist is in reality removed to a certain distance from the walls of the chest by the intervention of adventitious fluid or solid matter; in the former, no such removal of the pulmonary substance occurs.

Weak respiration depends on some obstruction to the entry of air into the part in which it exists; and is of variable seat, limited to a spot in one lung, or pervading the entire of both lungs.

The *superficial* variety occurs in the (*a*) *persistent* form in obstructive diseases of the larynx and some of the pharynx; narrowing or obliteration of a bronchus by contraction, thickening of mucous membrane, accumulated mucus, or pressure of tumours, cancerous or other; bronchitis; chronic consolidation of the lung, or infiltration with tubercle or other morbid product in a limited space; vesicular emphysema, pneumonia, previous to engorgement and after resolution; pulmonary œdema; pulmonary apoplexy, imperfect respiratory movements from paralysis. In the (*b*) *intermittent* form, it occurs in pleurodynia, in the dry and plastic stages of pleurisy, spasm of the glottis, spasmodic asthma, and where foreign bodies block up the air-passages. This intermittence constitutes an important feature in the physical signs of a foreign body in either bronchus, depending manifestly upon its motions from place to place, and consequent greater or less interference from time to time with the entry of the air. In some cases of pneumonia the same intermittent character of respiration may be observed; it indicates intermittent pressure on the main bronchus of the affected side.*

The *deep-seated* variety occurs in the effusion-period of pleurisy, in hydrothorax of moderate amount, and in pneumothorax.

(c) *Suppressed respiration* consists in a total absence of the respiratory murmurs, without their being replaced by any kind of audible phenomenon. There is a complete negation of breathing-sound in the part; but, it is urged by M. Fournet, there is still heard in some cases, towards the close of the movement of inspiration, a slight sound, "which appears to result from the lateral pressure, exercised on the pulmonary tissue, of the column of air which fails in entering the bronchial ramifications." For this sound he proposes the name of "sound of pulmonary compression." I have not, in such cases, been conscious of the recurrence of a distinct *sound;* but an indefinable sensation of struggle within the chest against obstruction may often be perceived.

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* Case of Beckett, U. C. H. July 1850.

The causes which produce weakness of respiration entail, when carried to extremes, its total suppression. It is probable that the respiratory murmurs are actually more or less extensively suppressed in almost all cases of infiltrations of the lung, tuberculous or other; but it is, for obvious reasons, in rare instances only that the suppression can be detected,—adjoining exaggerated respiration masks the local deficiency. In some rare cases of pneumonia (independently of accidental coexisting obstruction of a main bronchus) there may be no audible respiration,—I have seen two such; but, clinically, the common causes of such suppression are fluid and aeriform effusions in the pleura, and complete obstruction of a bronchus; in some rare cases of vesicular emphysema, and during intense paroxysms of spasmodic asthma, all respiration-sound may be locally deficient.

(β) *Changes of Rhythm,* without or with other modifications.

(d) *Jerking Respiration.* When the movement of inspiration, instead of being accompanied by a murmur continuous from the outset to the close (which may be represented thus ......), is attended with a sound of an interrupted character, divided into several unequal parts, (thus ... | .. | ... | ... &c.,) the respiration may be called *jerking.* The expiratory sound much more rarely possesses this peculiarity, and, as far as I have observed, is scarcely ever affected alone; but it is generally somewhat increased in duration, while the inspiratory (if the moments, during which sound is actually perceptible, be alone considered) is certainly somewhat decreased in this respect. The quality of the *inspiration* may have a tendency to harshness, and (by an exception to the general law) more than that of the *expiration.*

Jerking respiration may exist, through the entire of one or of both lungs, when it deserves the name of *general* (this is very rare); or be limited to a certain spot, when it may be called *partial*. The former variety is observed in incipient pleurisy, pleurodynia, hemiplegia, spinal irritation, hysteria and spasmodic affections of the air-passages. The partial variety occurs in lung-infiltrations, (most frequently the tuberculous,) and in cases of pleuritic adhesion.

(e) *Divided Respiration.* Instead of the two murmurs, inspiratory and expiratory, succeeding each other so closely in each act of respiration that they may be considered continuous, they are sometimes separated by a distinct interval or pause: to respiration of this rhythm the name of *divided* may be given. In advanced emphysema this separation between the two murmurs appears to depend on the destruction of the walls of the air-cells, and the impaired clasticity of the remaining texture. In all the varieties of bronchial and blowing respiration there is a pause, more or less marked, between the two sounds, the cause of which is not very clear.

(f) *Altered ratio of inspiration to expiration.* Instead of the former of these murmurs being to the latter (when this is audible at all) very closely, as 3 : 1, in point of intensity and duration, the ratio may be converted into as 1 : 4. This enormous relative prolongation of expiration is only met with in emphysema; but less degrees occur in various indurated conditions of the lungs, (especially the tuberculous,) and in pleuritic effusion and pneumo-thorax. I can scarcely believe that prolongation of the expiratory sound is capable of existing independently of other alterations, as I have never met with it to any well-marked amount without co-existing change of quality. However, in some cases, the increase of duration is much more marked, and therefore, clinically, more important than the change of quality, (in certain cases of tubercles, and of emphysema, for example).

The signification of the expiratory sound was, in some degree, mistaken by its re-discoverer, Jackson; he exaggerated its specific importance as a diagnostic sign of tubercles, and in this he has been very generally followed. Many persons forget that what may appear in a given individual, as compared with another, *prolonged* expiration, is really in him a natural state; some confound with it the pharyngeal expiratory sound; the normal existence of lengthened expiratory sound at the right apex of many females is habitually forgotten; and too few observers seem to be aware that, under whatever circumstances an obstruction exists to the free circulation of air in the lungs, the expiration will be prolonged—an obstruction which (experience shows) rarely, if ever, acts on the duration of the sound, without affecting some of its other properties. The fact of obstruction appears to me to account satisfactorily for the phenomenon. Expiration is (unless in exceptional cases) sonorous under all circumstances: where the progress outwards of the air is rendered more difficult and slower than natural, the most natural consequence in the world is, that the attendant sound should be proportionally intensified and lengthened.

Diminished elasticity of the lung will, if there be obstruction superadded, produce the maximum prolongation, as in emphysema with bronchitis; very often, however, the sound spoken of as prolonged expiration in this disease is nothing more than sibilant rhonchus,—true breathing sound being totally absent.

In some cases of incipient tuberculisation the inspiration seems to struggle against minute obstructions in the finer tubes, whence a rhythm and character of sound resembling that of a *cogged wheel* in rotation: it appears probable that glutinous mucus adherent to the walls (which the air disturbs, without forming into bubbles, as it passes) may be the cause of the phenomenon. I have observed it (and pointed it out in the clinical wards) repeatedly, and never but in the incipient stage of tubercle.

To the present head, also, belongs the intermittent weak respiration, described a moment since.

(γ) *Changes of Character, and in addition of other Properties.*—(g) In ***harsh respiration*** both murmurs have lost their natural *softness;* a peculiar *dryness* accompanies them; the breezy character of health is exchanged for one sharper and more blowing, which is generally more marked in expiration than inspiration. The *intensity* of the expiratory murmur appears augmented from this superadded character; its *duration* is increased. Both these latter properties may be, and commonly are, unaffected in the inspiratory sound. This type of respiration insensibly passes into *bronchial respiration*, a higher grade merely of the *harsh* type. Both murmurs are *rough* and *hard*, and notably more *dry* than in the natural state; the sharp blowing quality is heard in inspiration as distinctly as in expiration, and in the latter to a greater degree than in respiration of the previous type; the *intensity* of both sounds appears increased, the *duration* of expiration is considerably augmented, and is even greater than in normal bronchial respiration.

In harsh respiration the expiratory murmur commonly alone suffers change of quality, in the bronchial variety the inspiratory shares in the alteration. The usual earlier implication of the sound of expiration was first noticed by Jackson, and has since been insisted on by M. Fournet. This writer states that alterations in *character* always set in with the expiratory murmnr, and only affect the inspiration secondarily. With this statement I find that my own experience accords (if the word *habitually* be substituted for *always*) in respect of chronic maladies: I believe it to be correct also in the very great majority of cases of acute alteration of the parenchyma of the lungs, as in pneumonia. There is, however, a kind of rare bronchial respiration in which the inspiration alone suffers, the expiration retaining its natural properties; this seems to me insignificant in diagnosis, and rather to be an individual peculiarity of healthy respiration.

Harsh respiration, attending condensation or rarefaction of pulmonary substance, and dryness of the mucous membrane of the bronchi, is observed in incipient tuberculisation, dry bronchitis, vesicular emphysema, chronic pulmonary consolidation, dilatation of the bronchi, incipient cancerous infiltration of the lung, cases where the lungs are slightly compressed by plastic or tuberculous matter in the pleura, pneumonia, (period of resolution), various periods of pleurisy (in certain relationships to the effusion-fluid) and in pulmonary apoplexy. Bronchial respiration is met with under all circumstances of slight condensation of lung-substance, unless, between the condensed part and the surface, there exist such quantity of healthy tissue as to mask the morbid sounds by its own natural ones,—or, unless fluid, solid, or gaseous accumulation in the pleura interfere with the conduction of the sounds.

(h) In *blowing respiration* both murmurs are, as the name indicates, of blowing or whiffing character; and, in its most marked degrees, a sensation as if the air were drawn during inspiration from the observer's ear, or from the surface of the chest, and puffed back during expiration, is distinctly perceived. Both murmurs are continuous, *rougher* and *harder*, and especially *more dry* than in altered states of less-advanced type, and more or less *metallic*. The expiratory sound is of much *greater duration* than in the natural state: the inspiratory varies in this respect; both are more intense and of higher *pitch* than natural; and in both, *quickness* of production and progress constitutes (in the most marked forms especially) a characteristic feature.

There are three varieties of blowing respiration: (h. 1.) the simple; (h. 2.) the cavernous; (h. 3.) the amphoric.

(h. 1.) *Simple blowing respiration* is commonly associated by writers with the bronchial variety. This seems an injudicious attempt at simplification; for the two species not only differ in intensity, but in quality (mere bronchial respiration is never metallic), in quickness of evolution, and in pitch;—it seems, too, that the term bronchial should be limited to morbid respiration, simulating that naturally heard in the bronchial tubes.

Simple blowing respiration occurs in two forms,—the *diffused* and the *tubular*. If to the description above given be added the qualification that the whiffing murmurs appear to be produced with but moderate intensity, and sometimes at a distance from the ear, over a tolerably extended space, the description of the *diffused* form will be completed. In the *tubular* form, on the contrary, the phenomena appear to occur in a space limited to the immediate neighbourhood of the part examined, and that space to be of tubular shape, cylindrical or flattened. The metallic character is highly developed, to such a degree that the sounds may, without exaggeration, be compared to those produced by blowing sharply through a brass tube: their *dryness, rapidity of production* and *intensity* are still greater than in the *diffused* variety. It is in the tubular variety, too, that the sensation of air being drawn from, and puffed back towards, the ear is most distinctly marked.

Now the tubular form occurs in perfection in but one condition of lung, that of hepatisation; so true is this, that tubular and pneumonic breathing may be used as convertible phrases. But not unfrequently pneumonia runs its course without having produced true tubular breathing,—diffused blowing alone being audible. Now it appears probable (but I am unable to support the notion by reference to post-mortem examinations) that the *diffused* blowing respiration is transmitted from a number of small bronchi, the *tubular* from a few large ones. Were this the case, the change in character from *diffused* to *tubular blowing* would be referable to the compression and obliteration of the smaller bronchi, corresponding to the increase in extent and amount of solidification, and would account for the fact (which may, I think, be frequently observed) that the well-marked *tubular* form signifies a more advanced degree of disease than the *diffused:* great frequency of respiration tends also to give the sharpness of the former to the latter form. Less perfect tubular breathing is heard in cases of pulmonary abscess, or solid accumulation of moderate size, near the larger bronchi. The diffused form occurs, or may occur, in all conditions of diffused solid consolidation, intra or extra-pulmonary, simple, tuberculous, carcinomatous, fibro-fatty, &c., and in dilatation of the bronchi. There is, besides, a condition under which I have frequently observed the diffused variety of blowing respiration, and where it is likely, unless the examination be conducted carefully, to deceive the observer. In certain cases of tuberculous excavation, auscultation may be performed in two or three places without cavernous respiration with the hollow metallic character being discovered, the diffused variety of blowing respiration being the condition observed: were further investigation not made in such cases, the lung might be considered the seat of simple solidification. By moving the stethoscope carefully over the entire surface, cavernous respiration is usually detected, and the evidence of excavation established. In these cases the cavernous character is evidently masked by the diffused blowing, which is the result of the condensation existing around the excavation. This is one reason why a cavity may escape discovery, unless the examination of the chest be very carefully performed. On the other hand, in certain cases of pneumonia of the anterior apex, of tumor directly connected with a large bronchus, and of dilated bronchi, true blowing respiration may acquire a hollow character, and hence simulate cavernous breathing.

Lastly, in pleuritic effusion, if there be adhesion or agglutination of the pleura, respiration of the diffused blowing type (often sufficiently marked to suggest the idea of hepatisation) is more or less extensively audible; the presence of condensed lung near the surface sufficiently explains its existence. But it is not alone in these cases of adhesion, that blowing respiration may attend pleuritic effusion; it may be present where no adhesion exists, and the effusion is abundant. It does not, as far as I have seen, pass into the tubular variety under these circumstances, is never intense, and in the majority of cases, very slight (especially when compared with the amount of percussion-dulness present); it is also deep-seated, except in the close vicinity of the spine, and generally limited to the middle height of the back—in other words to the neighbourhood of the main bronchi: in some cases it has all the characters of, and is obviously, transmitted pharyngeal respiration.

The theory of bronchial (and blowing) respiration commonly admitted is, that the murmurs produced by the air moving in the larger bronchi, or (as the lung-movement is excessively diminished, and therefore little air—it may be none—can reach even the medium-sized tubes) the air, moving in the trachea, larynx, and main bronchi of the non-affected organ is transmitted thence through the hepatised or compressed tissue, with an intensity directly as the conducting power of the lung is increased by consolidation. Three objections occur to me as negativing this explanation: pneumonic murmurs habitually differ in quality and pitch from those of the trachea and larynx; they are habitually much stronger than the latter; while in some cases of perfect consolidation (without obstructed bronchus) there is dead silence over the hepatised structure. Skoda* holds that the laryngo-tracheal murmurs and those of both main bronchi consonate in the air contained within the bronchi permeating the consolidated parenchyma, and thus produce the phenomenon of bronchial respiration, when intense and of high pitch; whereas weak bronchial respiration of low pitch comes directly without consonance from the lower part of the trachea, the main bronchus, or one of those of second order. This essential distinction of the sources of weak and strong bronchial respiration seems artificial, and is not supported by clinical facts; besides, the pitch of metallic pneumonic respiration is irregularly higher than of the laryngo-tracheal sounds, whereas consonating sounds are always either of the same pitch, or of pitches bearing a certain definite relation to each other. The intensification of the respiration seems to me in the main a phenomenon of bronchial echo,—and its variability to depend on the state of the lining mucous membrane (tense and firm, or loose and flabby) being favourable or not to reflection of sound. On the same principle, elevation or depression of pitch becomes intelligible. The conducting power, in regard of breathing sounds, of hepatised tissue removed from the body, is found by experiment to vary inexplicably.

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* Auscultation, 4th ed. pp. 91 and 104.

(h. 2.) In the *cavernous variety* the ear receives the impression most distinctly of connection with an excavation of moderate size; the *character* of the murmurs is *hollow*, whiffing, and moderately metallic; their pitch lower than in tubular breathing. The peculiar *quickness* of production is less marked than in either kind of simple blowing respiration,—there seems to be a delay in the interior of the cavity. Cavernous respiration is rarely attended with any very notable degree of the sensation of air being drawn from the surface and puffed back again. This peculiarity does, however, sometimes exist, and appears to announce the close vicinity of the seat of the phenomenon to the surface of the chest.

The veiled puff (*souffle voilé*) is a modified cavernous respiration, in which a "sort of moveable veil interposed between the excavation and the ear" seems to be agitated to and fro; at least such is Laennec's description, but I do not profess to have any experience of the phenomenon.

Dilated bronchial tubes, and excavations in the substance of the lung (the less fluid they contain, the more solid their walls, and the freer their communication with the bronchi, the better) give cavernous respiration. Tuberculous excavation is by far its most frequent cause,—cavity produced by gangrene, abscess, cancer, pulmonary apoplexy, and intra-thoracic purulent collections perforating the lung, much rarer ones.

Cavernous respiration seems to be the neighbouring bronchial breathing modified by echo. Among the physical conditions most favourable to the perfect development of the different varieties of blowing respiration, is emptiness of the space in which they are actually produced, and from which they are transmitted to the ear. This is true of all the varieties; and hence true, whether the seat of the phenomenon be the bronchi, an excavation in the substance of the lung, or the pleural cavity communicating by a perforation with the bronchi. If the bronchi or pulmonary cavity contain fluid, the tendency will be to the production of various liquid rhonchi instead; and hence the phenomena, for example, of cavernous rhonchus and cavernous respiration are habitually in the inverse ratio of each other, as regards degree and perfection of development. If an excavation be partly filled with fluid and partly with air, the conditions observed are of three kinds: *a.* When the quantity of liquid is small, and the bronchus communicating with the excavation opens above the level of the liquid, pure cavernous respiration will be heard: *b.* When the communication with the bronchus occurs below the surface of the liquid, cavernous rhonchus is heard alone: *c.* When a double communication exists, that is, above and below the surfaces of the fluid, cavernous rhonchus and respiration will be both present. All this has been *observed* by myself and others.

(h. 3.) *The amphoric variety.*—The *special character* of this variety is derived from the sensation, which attends it, of air passing into a large empty cavity having dense walls—such as is perceived on blowing into a water-croft. It is strongly metallic, and sometimes, but rarely, *associated in one and the same respiration* with *metallic tinkling.* The amphoric character accompanies both sounds, but especially the expiratory, though occasionally most marked in inspiration; in some instances it only appears on forcible breathing: in rare cases it may be heard even at a distance from the chest.* Commonly audible at the middle height of the chest, rare at the upper part, and yet rarer at the base, its physical cause and theory are much the same as of cavernous breathing; the echoing cavity is simply larger. Hence it *may* exist in tuberculous or other excavations in the lung, but they are rarely of sufficient size; broncho-pleural fistula with hydro-pneumo-thorax is its common anatomical cause, whether produced (as is by far most usual) by tuberculous perforation of the lung and pleura, or by perforation of the lung attending the transit of pus from the pleural cavity (in perforating empyema) or from distant parts. The communication with the bronchi must, for perfection of the sign, be above the level of any fluid in the echoing cavity.

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* In a case by Louis (Phthisic, 2ème éd. p. 406), at 2¼ inches' distance.

A. 2. *Adventitious sounds, produced within the thorax by the act of breathing.* The essential difference between the morbid states of respiration hitherto considered, and the sounds now to be described, is, that in the former modifications only of a natural sound exist, while in the latter there is actual generation of new phenomena. These adventitious sounds may be produced in (*a*) The air passages, or cavities communicating with these; (*b*) The lung-substance; (*c*) The pleural cavities and mediastina.

(*a*) *Adventitious sounds originating in the air-passages* (*Rhonchi or rattles*).—A rhonchus may be defined as an adventitious sound, audible in inspiration or in expiration, or in both; of dry or humid character; masking the natural murmurs more or less completely; persistent or intermittent; originating in the air-cells, the minute or the larger bronchi, the trachea or larynx, and in excavations of the pulmonary substance; and produced either by the passage of air along bronchi of altered calibre, by air bubbling through fluid contained in the sites mentioned, or by the vibration of semi-solid plastic matter in the tubes, or of prominent folds of their own lining membrane.

Being unable to suggest any classification of rhonchi, at once demonstrably correct in principle, and clinically significant, I shall simply describe them one by one.

1. *Sibilant rhonchus*:—A dry-sounding rhonchus, of high pitch, of whistling character, commonly coexistent both with inspiration and expiration, but especially marked in the former, and occasionally limited to either, varying much in intensity and in duration, and recurring irregularly, instead of accompanying every respiratory movement.

Upon differences in duration mainly are founded two varieties, the short and the prolonged, or the *clicking* and the *whistling*. The former is a quick sharp sibilus, which ceases almost as soon as produced; the latter, a prolonged sibilation, of less sharpness, more slowly evolved, and sometimes lasting the entire length of the movement it accompanies. To these may be added the *hissing* variety, which imitates precisely that kind of sound.

Sibilant rhonchus is the index of bronchitis in some one or other of its various forms, or possibly of pressure on bronchial tubes. Laennec taught that the cause of sibilant rhonchus in acute bronchitis was alteration of the calibre of the bronchi, in consequence of the inflammatory thickening of their lining membrane and subjacent tissue. The natural intermittence of the rhonchus argues strongly against this notion, as the thickening of tissue must be a persistent state; and it is further opposed by the fact that either this variety of rhonchus or the sonorous may be interrupted and kept off for a variable number of respirations by causing the patient to cough. Coughing, we can readily understand, will alter the position of viscid mucus; but it cannot be supposed to have any direct and immediate influence on the thickness of the mucous membrane, or even on the conditions of a vibrating fold of its substance. The rhonchus (though dry to the ear) clearly depends on the influence of air on fluid, and may be imitated by blowing through saliva between the lips at a certain amount of separation.

When the cause (at least remote cause) of the sibilant rhonchus is of a permanent nature, as diminished calibre from the presence of a tumor, it is said that the rhonchus is itself permanent. My own experience does not support this statement. I have in certain cases of the kind discovered that the morbid sound will be altogether removed for a while by causing the patient to cough. This observation leads me to believe that in cases of the kind the altered form of the bronchus has less to do with the production of the rhonchus than the existence of local accumulation of viscid mucus, whether this be a mechanical result of the pressure of the tumor or the effect of circumscribed supersecretion. If this be correct, it is manifest that rhonchi of this class have no more claim in respect of their mechanism to the title of dry, than the crepitant or mucous: the impression made upon the ear alone justifies the application of the term.

There is no affection in which sibilant rhonchus is so marked as in pulmonary emphysema, and it is here particularly and exceptionally distinguished by its generally being of much more intensity and duration in expiration than in inspiration. In fact, when prolonged expiration is spoken of as a character of emphysema, the sibilant (or sonorous) rhonchus is often in reality referred to; for in aggravated cases this altogether supersedes (as far as hearing goes) the natural expiratory murmur: in such cases, indeed, respiration is little more than a continuous sibilant (or snoring) rhonchus. But it would be an error to suppose that the atrophy of the vesicles (or rarefaction of the lung), which fundamentally characterises emphysema, is the sole cause of the rhonchus. The various amounts to which the rhonchus exists in the same person at different times, the manifest increase in the phenomenon brought about by attacks of catarrh, seem to show most conclusively that attendant bronchitis is the most essential condition of its production. Perhaps, too, spasm has some influence on its development.

2. *Sonorous rhonchus* is a sound varying in special character, always marked by graveness of tone and dryness; usually coexisting both with inspiration and expiration, but especially marked in the latter, to which it may be limited; varying in intensity from a very slight sound to one loud enough to be audible at some distance from the chest, and to be attended with fremitus of the walls of the thorax over a surface of variable extent; varying in duration, but having a natural tendency to prolongation; continuously and steadily evolved unless of very short duration, when it is produced in a quick and abrupt manner; occurring interruptedly or attending every successive respiration (the latter is very rare); and sometimes alternating with the sibilant or with mucous rhonchus.

The special character of the sonorous rhonchus varies; hence arise a certain number of *varieties*. In the *snoring* variety, the sound is like the harsh full noise produced by a man snoring. When the sound is of short duration, rapidly and abruptly evolved, it resembles that produced by gently but quickly rubbing the finger, slightly wetted, upon a pane of glass, and may be called the *rubbing* variety: under other circumstances it resembles the musical and plaintive sound uttered by the wood-pigeon, and constitutes the *cooing* variety.

Like the sibilant, sonorous rhonchus suggests the ideas of vibration and dryness; both may be irregularly audible over the entire chest, or limited to one side; both are essentially of bronchitic origin. Sibilant and sonorous rhonchus are much less frequently heard in chronic than in acute bronchitis; and when they appear in the former, indicate a complication with acute irritation: in consequence of the less viscidity of the secreted matters in chronic cases, humid rhonchi supersede the dry.

As a general rule, the pitch of a dry rhonchus indicates its seat; the grave sonorous rhonchus originates in the large, the acute sibilant in the small, the very acute hissing in the minutest bronchi. But the calibre of a large bronchus may be accidentally reduced by pressure, thickening of its lining membrane, or the formation of a loose fold of this, especially at a point of bifurcation; hence practically sibilant rhonchus may and does occur in large-sized tubes. The more marked coexistence of sibilant rhonchus with either inspiration or expiration, has further been pointed out by M. Delaberge as distinctive of the size of the bronchi affected. If most marked in inspiration, the smaller bronchi are affected; if in expiration, the larger. This M. Fournet explains by supposing that the greater force of inspiration enables the air, during that movement, to cause the vibration of the smaller bronchi; while in the weaker expiration, force is wanting to produce the same effect: an explanation radically erroneous, seeing that, in point of fact, expiration is the more powerful act of the two.

M. Fournet describes a dry rhonchus produced in perfectly dry excavations in the lung, and differing from the common dry bronchial rhonchi simply by its cavernous quality, and by its always accompanying both respiratory movements. Of the occasional existence of this rhonchus in the site of empty cavities no doubt can be entertained; nevertheless I do not feel satisfied that the seat of this rhonchus is actually the cavity itself, and am rather disposed to believe that it is even under these circumstances a phenomenon originating in the bronchi, but modified in its character by the close vicinity of excavation. And, at all events, I do not believe that the sound could be produced in the cavity without the presence of some viscid material as a source of vibration, and which would necessarily deprive the cavity of the property of extreme dryness assigned to it by M. Fournet under these circumstances. The same remark applies to the sibilant or sonorous rhonchus which may similarly attend dilatation of the bronchi.

3. The *crepitant rhonchus* is of two kinds, primary and secondary:—a. *Primary*. This rhonchus, as its name indicates, suggests the idea of crepitation. When most perfectly developed, it occurs in puffs more or less prolonged, but rapidly evolved, composed of a variable, sometimes immense, number of sharp crackling sounds, all perfectly similar to each other ; conveying the notion of minute size ; dry ; coexisting exclusively, except in rare cases, with inspiration ; and once established, remaining a persistent condition until superseded by other phenomena.

The most accurate comparison which has been made between the crepitant rhonchus and any other species of sound seems to me unquestionably that of Dr. Williams, who compares it to the sound produced by rubbing slowly and firmly between the finger and thumb a lock of one's hair near the ear.. In every respect, both as regards the crepitations themselves and the entire act of crepitation, the similarity amounts almost to identity; but it must be remembered that it is to the perfect crepitation of primary pneumonia, and to this alone, that the comparison is to be understood to refer. Dryness is one of the most marked properties of true primary crepitation; and for this reason, among others, the common use of the term "bubbles," in speaking of the tiny cracklings which constitute the rhonchus, is extremely inappropriate. The sensation is not that of "bubbles" bursting; but rather of delicate tissue undergoing minutest ruptures with a crackling noise in many points simultaneously. Again, rapidity of evolution is an important property of the crepitant rhonchus, and among other characters serves to distinguish it from subcrepitation. The crepiti appear to be nevertheless successively produced in tissue nearer and nearer to the ear. So, too, persistency is a feature of some importance as distinctive of crepitant rhonchus: other rhonchi are manifestly influenced in the regularity of their production by the occurrence of expectoration, for example; but over true crepitation this appears to exercise no immediate control,—at least the rhonchus persists with all its characters as before, after the patient has relieved himself by expectoration. The first effect of a fit of coughing, indeed, is to render the rhonchus more distinct and abundant even than before; deep inspiration produces the same effect: under both circumstances parts of the lung, passive in ordinary breathing, are suddenly expanded.

When at its maximum the crepitant rhonchus accompanies the entire act of inspiration; when first developed, and when about to be superseded by blowing respiration, it appears towards the close of inspiration only. Under all circumstances it is, to say the least, rare to find this rhonchus co-existent in any degree with expiration; the statement that it may generally be heard to a diminished amount with this division of the respiratory act, appears to me to have originated in the confusion which long prevailed between the true crepitant rhonchus of pneumonia and the subcrepitant rhonchus of capillary bronchitis.

The crepitant rhonchus, thus characterised, is absolutely distinctive of the first, or congestive stage of pneumonia, and fixes its seat with precision; there are, certainly, rhonchi and pseudo-rhonchi which possess some of its properties, but none that possess them all combined. The mechanism of this rhonchus is yet undetermined, and its consideration implies that of certain phenomena themselves involved in obscurity, namely:—*a*. The intimate seat of production of the rhonchus; *b*. The physical condition of that seat at the moment of production. It has been said (*a*) the cavities of the pulmonary cells are the seat of the rhonchus; because:—1. The rhonchus is evidently a diminutive of the finer mucous bubbling species, which is confessedly produced in the smaller bronchi: the comparatively small size of the "bubbles" is explained by the less calibre of the cavities in which they are evolved. 2. The "bubbles" are extremely small and numerous, and equal in size, like the cells themselves. 3. In old people the "bubbles" are larger than in adults, just as the cells are of enlarged dimensions from the process of atrophy which the lung has undergone. 4. In infants, on the contrary, the "bubbles" are sometimes of extremely minute size, and in infancy the cells of the lung are well known to be proportionally small. 5. The shape of the "bubbles" appears to furnish an exact representation of that of the cells. But these arguments are not convincing. In the first, the question is begged; the difficulty is to ascertain whether the rhonchus *is* a diminutive of the mucous. 2. The sounds convey the notion of minute and of equal size, it is true; but this is no proof that they originate in the cavity of the cells. 3. The alleged fact is probably correct, and is connected no doubt with the atrophy which commonly exists in individuals of advanced age; but this atrophy may be understood to modify the rhonchus, without the seat of this being necessarily admitted to be the interior of the cells. 4. Against the correctness of this statement I must express myself most strongly. I have never yet heard a crepitant rhonchus having as much of the character of minuteness in the infant as in the adult individual; and, indeed, the non-development of the true primary rhonchus in children of very tender age is a fact familiar to all auscultators. 5. The notion respecting the "bubbles" seems to be an illusion; and its want of import is rendered sufficiently obvious by the circumstance that the best imitation of the crepitant rhonchus is furnished by rubbing a lock of hair firmly between the fingers. The real existence of "bubbles" is similarly shown to be, at the least, a matter by no means proved by the *character* of the rhonchus. For these reasons, I am disposed to believe that the development of the crepitant rhonchus in the *interior* of the vesicles may legitimately be questioned.

(*b*) The passage of air through liquid contained in the cells is commonly believed to be the physical cause of the rhonchus; but writers differ respecting the consistence of the fluid. 1. Some maintain that it is of aqueous or serous consistence, founding their belief upon the state of the liquid expressible from the lung after death during the first stage of pneumonia; and upon the fact that it is found experimentally, the more viscid the fluid through which air bubbles, the less perfect will be the similarity of the noise produced to the crepitant rhonchus, —to such a degree, that when pure mucilage is employed, no crepitant sound is produced at all. (Spittal.) 2. Others (for instance, Dr. Williams) maintain that the perfection of the crepitant character depends on the fluid through which air bubbles being viscid in a high or at least a notable degree. Each of these opinions is supported by experiments and arguments subversive of each other: but there is a more cogent argument against the notion that the consistence of the secretion occupying the cells exercises material influence upon the rhonchus; namely, that both the ordinary true crepitant and the exceptional subcrepitant rhonchi occurring in pneumonia coexist with expectoration of various degrees of liquidness or viscidity. Upon this point I perfectly agree with M. Grisolle, nor can I imagine that any one will be found ready to dispute the position.

It appears inferrible, from what has now been said, that the theories of the mechanism of the crepitant rhonchus hitherto proposed cannot be considered satisfactory: it is unfortunately easier to make this manifest, than to substitute a less objectionable one in their room. It seems, however, most probable, that the phenomenon occurs in the parenchyma of the lung itself, especially in those portions of it immediately contiguous to, and actually forming the walls of, the ultimate terminations of the bronchi; and that its physical cause is the sudden and forcible expansion of that parenchyma, glued together, as it were, by the viscid exudation with which it is infiltrated. Each single crepitus or click would thus signify the expansion of a cell, and be produced by the unfolding of surrounding glutinous tissue necessary for that expansion. Thus conceived, as respects its mechanism, the chief phenomena of the crepitant rhonchus become perfectly intelligible: its dryness and sharpness; the sensation of minute size attending the sounds of which it is composed; the similarity to the sound of minute ruptures of tissue, and the total absence of the bubbling character; its occasionally accompanying the entire inspiratory act, and sometimes appearing only at its close, according as the infiltration of viscid lymph less or more completely prevents expansion of the vesicles. We can on this supposition also readily understand why crepitation should exist in inspiration only: though the rapid and abrupt unfolding of the glutinous mass be productive of crackling noise, it is very unlikely that the comparatively slow and equable restoration of the tissue to its previous collapsed state would be thus productive,—indeed the presumed physical cause of crepitus has ceased to exist. On the other hand, there is no reason why in the ordinary theory crepitation should not as regularly exist, though not with the same *loudness*, in expiration as in inspiration. The air is presumed in inspiration to have passed through a certain fluid; if so, it must repass through it during expiration, and assuredly with a noise similar in kind, though less in degree: when rhonchi are manifestly produced by the passage of air through liquid (*e.g.* mucous, cavernous, &c.), they attend both inspiration and expiration. Further, the nature and mechanism of pleural, mediastinal and intra-parenchymatous pseudo-rhonchi support the views here taken.

b. *Redux.* The rhonchus crepitans redux, like its predecessor, suggests the idea of crepitation; but the crepiti constituting it are of more humid character and commonly more suggestive of "bubbling;" they convey the impression of larger size; are more slowly evolved, rarely, if ever, occurring in the abrupt puffs last noticed; are comparatively few in number; are more or less dissimilar to each other; somewhat irregular in occurrence; and frequently audible in expiration as well as in inspiration, though more specially appertaining to the latter.

Such are the ordinary characters of the rhonchus coexisting with the resolution of pneumonia. But in a certain number of cases its properties are much more similar to those of the primary variety: it possesses the same dryness, the same minuteness (I have never, however, observed redux crepitation of *greater* delicacy than the primary, as M. Grisolle appears to have done), and coexists with inspiration only. The observation of these facts, and of the vague manner in which the phrase "redux crepitation" is applied to the rhonchi existing in lungs undergoing the resolution of pneumonia, coupled with examinations made for the express elucidation of the point, has long led me to the conclusion that under that phrase are confounded two very different phenomena. These phenomena are a slightly modified subcrepitant rhonchus, and a true returning primary crepitation. The former is by far the more common, has all the characters of a humid rhonchus, and is, I can scarcely entertain a doubt, produced in the minute bronchial tubes; the latter, which affects the characters of primary crepitation, is probably generated in the same seat and manner as this. It will be observed that the rarity of true redux crepitation is in perfect accordance with the theory, just offered, of the primary rhonchus: it is in truth unlikely that the physical condition of the interstitial plastic exudation should often be similar at the two opposite periods of the malady, and hence improbable that a given phenomenon depending for its existence on that condition should frequently occur with identical characters at both those periods. But the thing may, *à priori*, be conceived a possible occurrence; and so we find by observation, that the effect which would follow, did it occur—that is, the re-appearance of the true primary rhonchus—*is* occasionally met with. On the other hand, were the primary rhonchus produced by bubbling in the interior of the vesicles, &c., as commonly supposed, there is no reason for imagining that the return of true primary crepitation should not be an invariable sequence of resolution.

The immediate cause of the subcrepitant rhonchus attending resolution is probably the bubbling of air through the fluid contained in the minute bronchi; and this fluid may be the result either of capillary bronchitis, or be merely on its passage from the previously engorged and now œdematous lung. This latter opinion seems calculated to throw some light upon the cause of a circumstance with which auscultators are well acquainted; namely, that the redux *sub*crepitation of pneumonia sometimes lasts but a few hours, and at other times persists for weeks.

4. *Subcrepitant Rhonchus.* Here the component crepitations have a distinctly bubbling character; they are of moderate size; humid; scarcely ever occur in puffs; are evolved with variable quickness, but rarely with much rapidity; are few in number and dissimilar to each other; occur with more or less irregularity; and attend both respiratory movements.

The different properties of this rhonchus are subject, within certain limits, to variation; hence arise varieties which are each distinctive of a separate malady, especially when the properties of the sounds are considered in connection with the seat of their production. (a.) In the *true subcrepitant*, the bubbles are of the smallest size observable in any variety of this rhonchus; their humid property is masked by viscidity; there is a certain tendency to occur in puffs, which are evolved with some abruptness; the bubbles are tolerably numerous and inclining to similarity in point of size; they occur with regularity, and are much more marked in inspiration than in expiration. (b.) In the *liquid* variety the bubbles are larger, distinctly liquid, and non-viscous; they do not occur in puffs, are of unequal size, recur irregularly, and attend expiration almost as abundantly and constantly as inspiration.

Produced by bubbling of air through liquid of variable consistence in minute bronchial tubes, this rhonchus, in the *true* form, if occurring at both bases posteriorly, indicates idiopathic capillary bronchitis,—if at either apex, tuberculous bronchitis,—if at one base posteriorly, it most commonly depends either on pneumonia in a state of resolution or on tuberculous disease of the *upper* lobe. In the liquid form it occurs in various parts of the chest from pulmonary apoplexy,—at both bases posteriorly from idiopathic or post-pneumonic œdema. Auscultators are indebted to M. Louis for clearly establishing the frequency and the pathognomonic character of subcrepitant rhonchus at both bases, as a sign of capillary bronchitis. It is now well understood that the marvellous success obtained by certain French physicians in the treatment of "pneumonia" depended on their having taken what was in reality capillary bronchitis for the former disease,—the immediate source of their error being confusion of the subcrepitant with the true crepitant rhonchus. The existence of the sound at *both* bases or at *one* base only is of great utility in distinguishing the two species of rhonchus. However, true subcrepitation may be found at one base only in cases of emphysematous bronchitis; though, when thus limited, it is generally connected, as shown by M. Louis, with tuberculous disease superiorly.

5. The *dry crackling rhonchus* is composed of a succession of minute, dry, short, sharp, crackling sounds, few in number, rarely exceeding three or four in a respiration; coexisting exclusively, or almost exclusively, with inspiration, though in very rare cases most obvious in expiration, especially when it has existed for some time; permanent, in the great majority of cases, after its characters have once been perfectly developed, until it ceases altogether to be produced, in consequence of its passing

into the humid crackling rhonchus; and usually conveying the impression to the ear of being evolved at a distance from the surface.

In stating that the dry crackling rhonchus, once *perfectly* developed, remains commonly a persistent condition until the transition into the *humid* form is established, I wish to lay particular stress on the circumstance of its being so developed. While yet producible only by forced respiration, and appearing only with an occasional inspiration, it is liable to disappear for a day or two, and then recur; but when once it has acquired sufficient perfection and stability to maintain itself steadily through a number of ordinary respirations, it apparently constitutes a persistent state.

This rhonchus, though its mechanism be unexplained, is of considerable diagnostic importance. It is of tolerably frequent appearance on the eve of the softening process in tubercles, and *may* hence be discovered wherever that product exists in the first stage. In the great majority of cases it is found in the infra-clavicular, or supra-scapular regions,—and most commonly in the former,—that is, when appearing *primarily*; it may form, *secondarily*, in a lower situation, when the disease has advanced to the second stage superiorly. It has not yet been detected as a primary condition in those rare cases in which tuberculisation commences inferiorly. Its normal course is to pass into humid crackling; and, according to M. Fournet, in cases of acute phthisis, the transformation occurs in the majority of instances in from eight to twenty days: in from twenty days to two months and a half, or three months, in the chronic form of the disease. These statements respecting time must be received with caution, however, and are subject (as admitted indeed by this writer himself) to very distinct exceptions.

6. *Humid Crackling.* A rhonchus composed of a series of crepitations, having a clicking character,—few in number,—of moderate size,—occurring during both respiratory movements, but with greater regularity and distinctness of character in inspi-

ration, and eventually passing into, or rather superseded by, the mucous species.

Although this rhonchus may be very satisfactorily connected clinically with incipient softening of tubercle, its mechanism is almost as obscure as that of the dry crackling. It appears rather to be produced in *direct* connection with tuberculous matter, which has commenced to undergo softening, than to constitute a mere form of bronchial rhonchus, having its seat in the bronchi, and produced by bubbling of air through mucus. The clicking character it possesses, combined with the special nature of the anatomical conditions which it is known to attend, points to a peculiarity in its mechanism. Possibly it originates in the interior of softening tubercles which have just commenced to communicate with the minute bronchi. If so, its "conversion" into mucous rhonchus would in reality be nothing more than its being superseded by that state.

7. *Mucous Rhonchus:* A rhonchus composed of bubbles of some size, considerably larger than those of the subcrepitant,—variable in number, and unequal in size,—distinctly liquid,—irregular in recurrence,—modified by the acts of coughing and of expectorating, and co-existing with both respiratory movements. The size of the bubbles localises the rhonchus (*mucous* and *submucous*) in the larger or smaller bronchi.

Caused by bubbling of air through liquid (mucus, blood, pus) contained in tubes of moderate or considerable calibre, and most commonly audible towards the central parts of the lungs, this rhonchus attends the secretion-stage of bronchitis, bronchorrhœa, dilatation of the bronchi, bronchial hæmorrhage, pulmonary apoplexy (with hæmoptysis), evacuation of pus from the pleura or elsewhere through the bronchi, and occasionally, the third stage of pneumonia.

8. *Cavernous* (or gurgling) *Rhonchus.* This rhonchus consists of a limited number of bubbles of large size, distinctly humid, occasionally disappearing for a time, having a peculiar ringing hollow metallic character, and coexisting commonly with inspi-

ration and expiration,—in some cases with either alone,—and attended or not with cavernous respiration. The size of the bubbles varies; whence the rhonchus has been called *cavernous* and *cavernulous*. In the latter there is distinctly a clear metallic character, but the sensation of hollowness is not perceived, or at least imperfectly. Cavernous rhonchus may be sometimes heard at a distance from the patient's chest, and the movement of the liquid perceived by placing the fingers on the spot, if the excavation be superficial.

The causes of temporary cessation of cavernous rhonchus are—1. Complete evacuation of the contents of the excavation; under these circumstances it is replaced by cavernous respiration; 2. Diminution of the contents to such extent as to bring the level of these below the bronchial opening or openings into the cavity; 3. Absence of air in the cavity, the entire space being filled with fluid; 4. Obstruction (by inspissated muco-pus or otherwise) of the bronchi communicating with the cavity; 5. Pressure of the lung by pleuritic effusion,* —a rare cause, seeing that the surface of the excavated parts is commonly agglutinated to the costal pleura.

Whenever bubbles burst in a hollow space within the chest, resulting clicks resound and are echoed by the walls of that space,—the harder and smoother the walls, the more perfect the echo; the softer and more anfractuous, the less perfect. So true is this, that excavations of small size, and surrounded with (what is very rare) healthy parenchyma, furnish scarcely any real cavernous rhonchus,—the bubbles form, but their clicks are not echoed. The common cause of bubbling is, of course, the passage of air in and out of the excavation; and, according to the relative position of the contained fluid and the communicating bronchi, will the period of the acts of respiration, at which the rhonchus is best heard, vary. The bubbles may sometimes probably burst at the bronchial orifices, and then echo within the cavity. Besides this, a species of gurgling, resembling cavernous rhonchus somewhat, (and becoming metallic in quality, if the mouth be held open,) may be produced in large cavities from the agitation of their contents by the impulse of the heart.* I have only heard this in the left lung, but Dr. Stokes has observed it in both, and even in the posterior portion of the right lung, producing a "tick loud enough to reckon the pulse by." Under all circumstances it is rare,—even when the heart and cavities are closely contiguous.

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* Case of Stafford, Consumption Hospital, Chelsea.

Excavations from tubercle are, of course, the most common anatomical state present; but those of abscess, sphacelus, softened cancer, pulmonary apoplexy, and perforating empyema, &c., as likewise dilatation of the bronchi, may produce the phenomenon. The cavernous rhonchus which occurs in the rare association of a circumscribed collection of pus in the pleura with bronchial fistula may, according to M. Chomel, be distinguished from that of an ordinary tuberculous cavity thus: in the latter, the cavernous rhonchus diminishes in proportion as the examination is made further away from its centre of production; in the case of the pleural cavity (for so it may be called), the gurgling produced inferiorly (the most common site of perforation) is propagated upwards, and exists with its full force within a limited extent around.

9. *Dry crepitant rhonchus with large bubbles* (*Laennec's Râle crepitant sec à grosses bulles*), "observed only during inspiration, conveys the impression as of air entering and distending lungs which had been dried, and of which the cells had been very unequally dilated, and entirely resembles the sound produced by blowing into a dried bladder." Such is Laennec's description of a rhonchus which he believed pathognomonic of emphysema, pulmonary and interlobular; he was probably right, but the phenomenon is excessively rare. It mainly indicates the existence of dry distended pouches under the pulmonary pleura,

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* Case of Jameson, U. C. H., males.

—the most advanced form of vesicular empyema. In the few instances in which I have heard it, it was transitory.*

(*b*) *Adventitious sounds originating in the lung-substance* (*pseudo-rhonchi*). If individuals, whose lungs are healthy, or diseased only at the apices, and whose breathing is habitually calm, are made suddenly to respire deeply, a peculiar, fine, dry crepitation, accompanying inspiration only, may often be detected at the bases posteriorly. But after two or three, or at most five or six, acts of respiration, it totally disappears. This pseudo-rhonchal sound seems to depend on the sudden and forced unfolding of air-cells, which are unaffected by the calm breathing habitual to the individual; and its only importance arises from the possibility of confounding it with crepitant rhonchus.

Here we have a minor degree of the phenomenon observable in the same regions, when any considerable portion of the base of the lung is under the influence of persistent pressure from tumors or enlarged abdominal organs,—I mean the pulmonary pseudo-rhonchus,† which consists of a series of fine, very dry crepiti, evolved at a peculiarly slow and drawling pace, variable in number, but generally very numerous, and commencing towards the close of inspiration, or in some cases apparently when this movement has almost ceased.

This sound of unfolding lung varies in quality, if the lung be diseased. Thus, in a case of arrested phthisis, at the apex of the lung, (where, nine months before, softening-signs were most manifest,) I have recently found dulness under percussion, feeble bronchial breathing, and a deep-seated creaking sound accompanying inspiration: in all probability the unfolding of induration-matter in the lung is the cause of this sound. I have repeatedly observed this sign, without having traced the progress of its development so well as in this instance. A faint creaking sound is sometimes audible in the same situation, which disappears after a few brisk movements of the corresponding arm, and is probably without importance.

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* Case of Hayes, U. C. H., July 1850.

† Clinical Lectures, loc. cit. p. 525.

(c) *Adventitious Sounds in the Pleuræ and Mediastina.*

I. *Pleuræ. Friction-sounds and pseudo-rhonchi.* — Daily experience proves that the collision of the opposite laminæ of each pleura, during inspiration and expiration, is not in the healthy state productive of appreciable sound. Experiments upon the lower animals might, were this necessary, be referred to in confirmation of the fact. This noiselessness of movement of the pleural surfaces upon each other depends upon their perfect smoothness and slight humidity; when these conditions become changed by disease, this gliding motion is attended by different modifications of sound, varying with the nature and amount of the existing anatomical change. While these different sounds all agree in being produced by friction, some of them, also, convey this sensation to the observer, and are, therefore, commonly designated as *friction-sounds.*

A. *Pleural friction-sound* consists either of a single, or, more commonly, of a series of abrupt jerking sounds, few in number, and manifestly superficial in seat: it is audible over a variable, but usually limited, extent of surface; persistent or intermittent; of variable, but commonly more or less considerable duration; varying, in point of intensity, from a scarcely audible noise to one of extreme loudness; attended with a sensation of dryness; almost invariably heard in inspiration, and habitually, but by no means always, more intensely developed with that movement; most frequently accompanying both inspiration and expiration, seldom (though more frequently than is commonly believed) expiration alone;* produced with ordinary respiration, or developed only after coughing, or by deep inspiration; and in strongly-marked cases attended with fremitus palpable to the hand, and perceptible to the patient.

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* Case of Clancy, U. C. H., April 3, 1849. Such total limitation to expiration is, however, of temporary duration.

The modifications of intensity and special character of friction-sound justify the establishment of four varieties: (a) *grazing;* (b) *rubbing;* (c) *grating;* (d) *creaking;* peculiarity of rhythm founds another variety, the (e) *rumbling.*

(a) *The grazing variety*, the most delicate form of friction, is usually a single sound; audible over a very limited extent of surface; occurring with an occasional respiration only; remarkable for mobility; more rapidly evolved, and of less duration than the other varieties; attended with a sensation of dryness, and limited strictly to inspiration. Varying from hour to hour, it may be, in precise site, this variety is almost peculiar to the dry period of pleurisy, (occurring rarely at the absorption period,) and is mostly met with in the phthisical form of that inflammation. In primary idiopathic pleuritis it is very rarely to be detected, because the period of its existence has usually passed away before the chest is submitted to examination. In cases of intercurrent pleurisy, where the patient has been under treatment for the primary disease, a better opportunity is afforded of establishing its existence. Another source of difficulty in its detection, even in tuberculous cases, is the shortness of its duration: a single day suffices for its production, development, and termination; and this series of changes may, as I have, though rarely, observed, be accomplished several times successively in the course of a few days.

It has been stated by M. Fournet, that the grazing sound is most commonly encountered at the upper parts of the chest, and indicates the progress of tuberculisation to the periphery of the lung. According to my experience, its ordinary seats are the infra-mammary, infra-axillary, and infra-scapular regions; I have not yet satisfied myself of its existence, either in the supra-spinata, supra-clavicular, or infra-clavicular regions: probably it is masked in these vicinities by the louder morbid sounds usually present. The sign is not only of clinical but of pathological importance, because it has helped to connect the fugitive chest-pains of phthisis with local and evanescent pleurisy; but my observation on the localities it occupies would lead to the inference that the cause of that pleurisy, though a dependence on the co-existing tuberculous disease, is not a direct consequence of the mechanical or vital irritation of the foreign matter,—is not usually dependent on actual extension of the tuberculous disease to the periphery of the lung.

(b) *The rubbing variety*, a more advanced type of friction, consists of a series of jerking sounds, rarely exceeding three or four in number; is audible over a tolerable extent of surface, provided the necessary condition of motion of the lung exist; of rather considerable duration, slowly evolved, attends both inspiration and expiration, and is more frequently than other varieties attended with friction-fremitus. It occurs in pleurisy, at the periods of plastic exudation, and at that of absorption (redux friction), being more frequently *observed* at the latter. The mechanism of friction-sound causes a difficulty in conceiving its production, where great liquid effusion is present in the pleura; and ordinary experience is in accordance with *à priori* considerations—the two conditions are not *observed* to coexist. Dr. Stokes was, however, the first to mention a case in which, though great and universal dulness of the side existed, friction phenomena were audible, and even perceptible to the patient, in the postero-inferior and lateral portions of the chest: "they may then," he infers, "coexist with extensive liquid effusion." There is one source of fallacy in such cases; supposing them to have reached the period of absorption, the fluid might have been nearly removed, and yet the condensation of the superficial strata of the lung, combined with an accumulation of pseudo-membranous matter, been sufficient to produce extensive and marked dulness. Now, under such circumstances, the production of friction-phenomena would have been inevitable. However, the retention of some portion of the lung's surface in tolerably close proximity to the costal pleura, by means of adhesions, also renders the production of friction-sound possible, although a considerable quantity of fluid be present in the pleura. M. Fournet states that he once ascertained the coexistence of these three conditions; and I presume that three examples I have myself observed of coexistent friction-sound, and general effusion-signs were thus explicable. Effusion-signs may be evident in the back, and friction-signs in front; this is common.

Laennec described friction-sound as an attendant upon, and one of the most important diagnostic signs of, interlobular emphysema; while he made no mention of its existence in pleurisy. The experience of his followers has reversed the connection which Laennec sought to establish: it is now believed that emphysema of any anatomical form is as incapable of producing friction-signs, as pleurisy is indubitably their common cause. Andral, Louis, Stokes, Fournet, and other writers, agree in denying, either directly or by inference, that the collision of subpleural vesicles, or of pulmonary septa rendered prominent by infiltration of air, against the opposite pleura, is an observed cause of friction-sound. Such was the opinion which I held upon the point also; and I still believe that we want the anatomical proof of the phenomenon being thus generated. But from some cases I have met with of very advanced emphysema, manifestly attended with a low degree of rubbing-sound at the postero-inferior part of the chest (where subpleural vesicles are very commonly developed), and presenting neither signs nor symptoms indicative of pleurisy, I am induced to think that Laennec's belief respecting the occurrence of friction-signs in some forms of emphysema was not erroneous.* Upon referring to certain cases in my possession of individuals dying with extensive infiltration of air under the pleura, I find the existence of friction-sound during life noted, and certainly not a syllable respecting false membrane in the pleura among the details of the post-mortem examination. I can scarcely think that so obvious an appearance as this would have been forgotten had it existed. These cases, too, are perhaps deserving attention, because collected at a period when I had a preconceived notion against the possibility of friction-sounds being evolved in the manner now referred to. It is true, Dr. Stokes maintains their evolution under the circumstances to be physically *impossible.* "It is only," he observes, "when the surfaces are rendered dry by an arrest of secretion, or roughened by the effusion of lymph, that their motions produce sounds perceptible to the ear." But this remark, which is perfectly just, if applied to these surfaces when holding to each other their natural relation of simple approximation without mutual pressure, begs the question at issue when applied to pleural laminæ, one of which presents elevations on its surface necessarily productive of some slight pressure against the other.

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* I have recently (case of Hayes, U. C. H., July, 1850, Case-books, Fraales, v[illegible]v. p. 25.) distinctly observed this in the mammary region.

(c) *The grating variety* conveys the sensation indicated by its name; and, except in respect of its greater sharpness, is characterised as the last variety. It may occur at the period of absorption, (with or without retraction of the chest,) and occasionally that of exudation. It sometimes indicates that the exudation-matter is laid down in the granular form; and (the physical reason is here much the same) may be produced by sub-pleural miliary tubercles, causing superficial prominences.

(d) *The creaking variety* is suggestive of the noise produced by the creaking of new leather of moderate hardness; in other respects it resembles the rubbing variety, with which it has a tendency to co-exist. It indicates dryness, firmness, and toughness, on the part of the pseudo-membranous matter, and hardly occurs, except in cases lapsing into the chronic form. The capsule of induration-matter, that sometimes caps tuberculised apices, furnishes in rare instances, either by its own motion on itself, or by collision with the costal pleura, a modification of this sound, most frequently audible in the supra-spinata fossa.

(e) Instead of being composed of a series of distinct interrupted jerks, friction-sound may be prolonged without cessation from the beginning of inspiration to the end of expiration; or this variety (evidently allied to pleural pseudo-rhonchus), the name of *rumbling* may be chosen.*

The practice of hospitals more especially teaches that, while pleurisy is among the more common of acute diseases, friction-signs are far from remarkable for frequency of occurrence, or at least of discovery. The following seem the chief reasons of this comparative rarity:—1. Liquid effusion generally occurs with great rapidity; the time during which friction-signs are audible has therefore frequently passed by when patients apply for medical aid;—2. Friction-signs may exist, but escape attention from their slight degree of development;—3. Or from auscultation not being practised immediately over their seat of production;—4. Or from too long a period being allowed to elapse between successive examinations of the chest;—5. In cases of absorption of pleuritic effusion, the development of redux-friction sound will be prevented if the two pleural surfaces be *agglutinated* together; because locomotion of the lung is thus prevented;—6. In all cases of pleuro-pneumonia in which, while the infiltration and enlargement of the lung are sufficiently great to prevent its expansion, resolution of the pleurisy occurs before that of the pneumonia, pleuritic friction-sound (if we believe M. Fournet) cannot be developed. Dr. Stokes has also expressly noticed the rarity of friction-phenomena in pneumonia: "in no case has he found them after hepatisation had formed; and their co-existence with the crepitating râle in the early stages is extremely rare." My observation is somewhat at variance with this statement: friction-sounds are occasionally audible directly over hepatised lung, if the consolidation be confined either to the centre or the periphery of the organ; besides, they are *often well marked* in front about the nipple, while the signs of hepatisation are perfect behind;—7. In some cases, friction-phenomena are audible in pleurisy after deep inspiration, when perfectly imperceptible during ordinary breathing. The natural inclination of pleuritic patients to restrain the motions of the chest as much as possible, tends to deceive the observer as to the non-existence of friction-sounds.

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* Case of Luckett. Clin. Lectures, loc. cit. p. 390.

The duration of each jerk of friction-sound, as also of the whole series in each act of respiration, depends on the extent of surface in a state to furnish the sound, the freedom of motion, and dilatibility of the lung. The length of time, friction-sounds may continue audible in a case of pleurisy, varies greatly.

The motion of respiration is, of course, the ordinary cause of the collision of surfaces required for the generation of pleural friction-sound; but (as first noticed by Dr. Stokes) the heart's impulse may, under favouring circumstances, produce it. The rhythm of ordinary pleural friction coincides with the movements of respiration; that of the variety, generated by cardiac impulse, agrees with the heart's movements; hence a difficulty (which is considered in Chapter II.) in determining on the pericardial or really pleural origin of such friction.

B. *Pleural Pseudo-ronchi.*—In the winter of 1842, I made the following observation:—In a male adult presenting the most evident signs, both in front and behind, of a cavity at the left apex, an extremely abundant subcrepitant rhonchus occurring almost in puffs, and having the liquid character in a most marked manner, was day after day (during the week previous to death) detected in the entire height of the left side posteriorly. The rhonchus was, however, distinctly more abundant and more liquid (as noted in writing during life) in the upper scapular and upper part of the lower scapular regions, than elsewhere. As the patient was anasarcous to a high degree (the urine albuminous), and as he constantly lay on the left side, the explanation of the subcrepitant rhonchus naturally suggesting itself was, that it depended on œdema of the pulmonary tissue generally, but most marked at the apex, and there of course affecting tissue between the cavity and the surface of the lung. At the post-mortem examination, however, I found this explanation was inadmissible; for the thin lamella of tissue between the cavity and the surface was as hard as cartilage, and contained not a particle of serosity; nor was the organ in any part distinctly infiltrated with fluid, being, on the contrary, particularly dry from its excessive induration. But all along the posterior surface of the pulmonary pleura there appeared (in addition to ordinary dense pseudo-membrane) a quantity of fine adventitious cellular tissue, abundantly infiltrated with liquid. Masses of some size were formed from place to place by the accumulation of fluid in the meshes of this cellular tissue, and it was observed by those present (who had not seen the patient during life) that they were much larger than elsewhere at the apex. There was no air either in the cavity of the pleura, or intermixed with the serosity. Now, although it was possible to suggest another explanation, it seemed most reasonable to suppose, under the circumstances, that the rhonchal sound was actually produced in the masses of infiltrated tissue referred to, and therefore outside the lung, and independently of air.

Subsequent experience has amply proved the correctness of this explanation, and shown that sounds, perfectly rhonchal in properties, are producible whenever adventitious tissue within the pleura is infiltrated with serosity, and the movements of the chest are sufficiently free. The sounds occur in two forms, *squashy* and *crackling*. The character of the first is represented by its name, and, coupled with the sensation of extremely superficial site, suffices for its diagnosis.* The crackling form, in itself indistinguishable from some conditions of subcrepitant rhonchus, may be diagnosticated by the co-existence of friction-sounds, constant or occasional, and by its being unaffected by coughing. Mere moisture in the plastic matter within the pleura seems enough to give a rhonchoid character to friction-sound.

II. *Mediastina.* Crepitation, inspiratory and expiratory, of variable degrees of dryness, abundance, and size, audible in forced respiration only, or in calm breathing, constant or intermittent, disappearing after a few chest-expansions, or continuing through a long examination, is sometimes to be discovered over the sternum, generally or partially, while it is completely wanting over the contiguous portions of lung. No symptoms of any kind necessarily attend this state.*

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* The *precise spot* where infiltrated false membrane exists may be pointed out during life by this sound: *e.g.* case of Griffiths, U. C. H., Males, vol. ii., p. 183. Feb. 1847.

How is it produced? When the cellular tissue is infiltrated with serosity and air, the production of such sound through the movements of the chest is quite intelligible. I observed the fact in a remarkable case, where air was infiltrated into the mediastina, through perforation of the œsophagus and pericardium, effected in the attempt to swallow a knife.† But whether the presence of air be absolutely requisite, I do not know as matter of experience. This pseudo-rhonchus derives its clinical interest from the likelihood of its being mistaken (as it has actually been) for the crepitant rhonchus of marginal pneumonia.

B. *Modified Resonance of the Voice.*—The signs derived from modified vocal resonance are uncertain in character and obscure in theory, and, though occasionally of important clinical signification, hold, as a rule, a low place among physical aids to diagnosis.

The natural vocal resonance may be (*a*) diminished in intensity; (*b*) increased in intensity, without or with alteration in quality.

(*a*) Diminution of resonance varies between weakness and suppression. *Weak resonance,* as its name implies, signifies a state in which the vocal resonance is distinctly less marked than natural; commonly spoken of as rare, it is really of frequent occurrence. Thus in the dilatation-period of liquid and aeriform effusion into the pleura, in cases of obstructed bronchus (main), even in solidification of the lung, and when excavations have formed, the resonance directly over these may be temporarily weaker than natural.

Suppressed resonance means that rare condition in which all transmission of the voice to parts of the surface, where naturally perceptible, has ceased; no audible sound being conveyed there. Impairment of vocal resonance exists in emphysema; and in pneumothorax (simple or fistulous) actual suppression may occur. But neither of these conditions is constant: in emphysema the resonance (probably from the influence of thickened and enlarged bronchi) may be bronchophonic. Simple pneumothorax is so rare, that we have little opportunity of substantiating its signs, and special peculiarities occur in the fistulous variety. During the dilatation-period of pleuritic effusion the resonance may be weakened even to suppression.

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* Case of Scott, U. C. H., July 23, 1850.

† Univ. Coll. Mus., No. 3859.

(*b*) Increased resonance presents itself in the forms of *exaggerated resonance* and *bronchophony*, which are little more than degrees of each other. The amount of resonance in the former undergoes simple increase; in the latter there is, besides, a greater concentration of sound: the former may be considered a *diffused* bronchophony; the latter a *concentrated* resonance of exaggerated force. In bronchophony the sound is also clearer, and more distinct; but still always unattended with articulation, and rarely producing any tactile sensation in the ear of the observer. Exaggerated resonance is a mere effect of conduction; bronchophony, as we shall by and by see, something more than this. The latter is usually a persistent phenomenon, so long as it exists; that is, produced every time the patient speaks. The space in which it is discoverable is frequently rather sharply defined; in other words, the transition from the most distinct bronchophony to the natural resonance is sudden; more commonly still a gradual diminution of the phenomenon is traceable. In quality it may be *metallic*, *sniffling*, or partake (as is most common in aged persons) of the tremulous and bleating tone especially characterising ægophony. Commonly ceasing abruptly, it sometimes fines off with an echoing character. As in the case of natural resonance, there is no uniform ratio between the strength of bronchophony and of tactile fremitus.

The physical conditions under which bronchophony is observed are: increased density of the pulmonary tissue surrounding pervious bronchi, whether that increase be from liquid, semi-solid, or solid infiltration, or from compression; presence of an indurated adventitious mass in the same situation;—and enlarged caliber, and hypertrophy of the substance of the bronchi.

In its maximum degree, and marked by the metallic and sniffling quality, bronchophony is heard coexistent with tubular blowing respiration, in the parts corresponding to the hepatised lung. In the simple forms it is heard in tuberculous and plastic consolidations, in pulmonary apoplexy, slightly in pulmonary œdema, and under certain conditions (*e. g.* when the new product and the bronchi are connected together) in cases of tumor within the chest. It exists in dilatation of the bronchi; but the enlarged caliber of the tubes is not habitually the sole condition of its presence; co-existing thickening, and hardening of their walls, and condensation, attended with chronic induration of more or less pulmonary substance around, contribute materially to its production. Still, increased width may be the sole appreciable cause of the sign. Bronchophony cannot be regarded as an ordinary sign of pleurisy with effusion. At the stage of effusion with general dilatation of the chest (a *broad* mass of fluid intervening between the lung and parietes), all vocal resonance has ceased; but in the two earlier stages of effusion, the laminar and the gravitating, this phenomenon may be detected under certain circumstances. Thus, in the immediate vicinity of the larger bronchi between the scapulæ, inside the ordinary site of ægophony, the resonance is generally bronchophonic. So, too, if there be induration of pulmonary substance (superadded to the mere condensation from pressure), bronchophony may be sometimes very manifest posteriorly and laterally; for example, where slight hepatisation coexists with effusion. Bronchophony may often be heard in the upper front regions, when the effusion is sufficient to condense a considerable portion of the lung inferiorly. Again, it may be detected (as long since shown by M. Reynaud) in a part of the lung which had just given ægophonic resonance, by causing the patient to alter his position in such manner as to displace the pleural fluid from the spot under examination. It is common, also, at the period of absorption and retraction of the chest-walls; and from the existence of partial pleural agglutinations or adhesions, bronchophony is sometimes locally audible throughout the entire course of effusion. This sort of resonance may also be the sole one audible over cavities in the lung. Compressed texture (whatever be the cause of compression) may give bronchophonic resonance, if of any notable bulk.

Laennec supposed the bronchophony of hepatisation explicable by the increased conducting power of the infiltrated and solidified parenchyma. Dr. Skoda, holding the general doctrine that "the varying conducting power of the healthy and diseased lung-structure cannot be taken as a basis of explanation of the phenomena of auscultation," opposes Laennec's views on the mechanism of bronchophony in particular, on the following grounds: that, first, bronchophony may in the course of a few minutes appear and disappear over hepatised lung, the other physical signs (especially those of percussion) having undergone no change; secondly, when vocal resonance has temporarily disappeared, it may be restored by making the patient cough or breathe deeply, so as (it is postulated) to free the bronchi in the hepatised part; thirdly, in cases of pleuritic effusion, the resonance weakens in proportion as the fluid (and *pro tanto* the solidity of the lung) increases; fourthly, if a healthy and a hepatised lung be taken from the body, and while one person speaks through a stethoscope placed in contact with the surface of each organ successively, a second listens through another stethoscope, it will be found that more intense resonance reaches the ear through the healthy than the diseased lung. Dr. Skoda supposes that bronchophony is really produced by consonance of the air, within the bronchial tubes, with the laryngeal voice; hence its occasional cessation would be explicable by the interference offered by mucus or other secretions in the tubes to the occurrence of consonance. He excludes the walls of the trachea and bronchi from all share in the conduction of the sonorous vibrations of the chordæ vocales, maintaining that this is the office of their contained air alone.

The results I have obtained from some experiments on the conducting powers of hepatised tissue, do not agree with those announced by Dr. Skoda. It is quite true that tissue, called hepatised, may conduct the voice no better, or even less forcibly, than a similar thickness of healthy parenchyma; but it is equally true that this is not a constant result. I have occasionally found hepatised lungs, taken from the body, conduct the sound with extreme intensity. And these varying results may be obtained from different lungs, which the naked eye would judge to be in the same state physically, in regard of their shares of air, fluid, and semi-plastic substance. But it is evident that, acoustically, they are in different physical states; and that, therefore, such experiments as Dr. Skoda's are not to be trusted to. Specimens of parenchyma, apparently identical, are in reality widely different. In estimating the conducting power of lungs, there can be little doubt that varying homogeneousness is a more important element than any observable so-called solidification in pneumonia. In the varying homogeneousness of different specimens may be the key to the difficulty; but even if so, it is a key which cannot practically be used. Again, if, while one person speaks into a stethoscope with its narrow end introduced into the trachea, a second listens over a part of the chest where hepatised lung lies beneath (and where intense sniffling bronchophony existed during life), the auscultator will often be surprised at the singular and total absence of sound. Dr. Skoda, obliged to admit this fact, attempts to evade its force by supposing the vibrations to be interfered with by fluid in the bronchi. To this I would reply, that I have established the total absence of post-mortem resonance over pneumonic solidification, where the bronchi, to the third and fourth divisions, were *peculiarly free from fluid*, and scarcely any spumous liquid infiltrated the parenchyma,—which very same parenchyma, removed from the body, conducted the voice from one stethoscope through another with striking intensity. If we consider the main difference in the physical conditions of the parts, when an individual himself speaks, or when another speaks into his trachea after his death, an obvious explanation of the experimental failures to imitate the bronchophony of life, suggests itself. In the dead body, in truth, the laryngeal and tracheal walls take no part in the production or conduction of the sound, which is propagated by their contained air alone; whereas in life the walls of those tubes obviously conduct the sonorous vibrations. But to admit this would have been fatal to more than one of Dr. Skoda's hypotheses. Besides, hepatised and healthy lungs are not strictly comparable in and out of the body in regard of this matter. *Within* the body the contact of a hepatised lung with the chest-wall is more perfect than of a healthy one; and, admitting that the former is a worse conductor in regard of the condition of its substance, it may be a much better one through *the closeness of its union* (*especially if adhesive*) *with the parietes.*

In seeking for a theory of bronchophony, there are three points especially to be considered: the conduction of laryngeal voice, its possible intensification within the chest, and the distance at which that intensification (if real) occurs from the part of the chest-walls examined. In regard of conduction, theory would say that as the human voice is best propagated in air, the more the lungs were rarefied, the higher would its conducting power become; and, in accordance with this, it is certain that intense bronchophony is sometimes heard over highly emphysematous tissue. But, on the other hand, as the tracheal and bronchial walls themselves vibrate during speaking, any *really solid* material directly connecting a large bronchus with the surface of the chest, must conduct those vibrations forcibly; and, in accordance with this, we find that wherever solid fibrous structure is seated in the manner supposed, bronchophony of the most intense character is audible. But if the union of the solid material with the bronchus on the one hand, and the chest-wall on the other, be not perfect—if there be any interruption at the planes of union of the conducting materials—the acoustic conditions are completely changed, inasmuch as interruption at the union of media of different densities most deeply impairs the conducting faculty of the series. Here is one clue to the differences in vocal resonance, observed in cases where the physical conditions appear, on superficial view, identical. On the other hand, theory, for the reasons just now adduced, does not teach that in the variable semi-solid states comprised under the title of "hepatisation," conducting power should be affected in a uniform manner, and experiment shows that it actually is not. Experiment, in truth, alone can teach what the force of conduction really is in the various complicated conditions of physical change in the lungs.

Next, in regard of intensification within the thorax. The thoracic resonance is sometimes stronger than the laryngeal voice; hence intensification of sound within the chest is real. How is it to be explained? Skoda's theory of consonance[*] does not seem to meet the difficulty, or rather to go beyond it. For, in the first place, the air in any enclosed space does not consonate with every sound produced at its orifice, but only with the fundamental note of that space, or certain others having a fixed harmonical relationship to that fundamental note.[†] Now, when well-marked bronchophony exists, it is audible, as I have assured myself, with the various notes of the octave, though most with the low ones. Again, bodies consonate only in unison, or in certain fixed harmony, with the original sound. Now, the pitch of the bronchophonic voice varies irregularly from that of the laryngeal, with which it co-exists. Further, it seems doubtful whether consonance could occur in a system of branching tubes, such as the bronchi; and lastly, Dr. Skoda's exclusion of the tracheal and bronchial walls from all participation in the phenomenon is at variance both with theory and experiment. But there is another way in which sounds may be intensified at a greater or less distance from their place of production—by reflection, and by reflections brought to a focus, or *echo.* Now, the conditions of reflection are fulfilled in the hepatised lung; the tubes along which the voice is transmitted from the larynx are surrounded by semi-solid material, proper (when compared with healthy tissue) to reflect and concentrate the sound; the air-cells and minute bronchi are closed to a variable distance, and prevent its divergence. The tubes resemble so many speaking-trumpets, and, just as in these instruments, the augmentation of sound is produced by reflection from their quivering walls; as this reflection tends to propagate vibrations (otherwise divergent) in the same direction, intensification of sound must be the result. But if the reflected vibrations chance to be brought to a focus within a large tube, then echo will occur, and, as under ordinary circumstances, the echoed may be materially louder than the original sound. But, it may be inquired, how, upon this theory, is the temporary disappearance of bronchophony explicable? In the first place, I disagree with Skoda as to the frequency of the occurrence; I believe intermittent bronchophony to be a most rare phenomenon,—in this respect assimilable to intermittent bronchial respiration. In the second, where it does occur, it may depend on the deadening influence of mucus and blood in the tubes,* or on obstruction of a chief bronchus by fluid, or by accidental pressure (glandular or other) on its external surface.

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[*] When vibrating sonorous bodies communicate their sonorous vibrations by sympathy to other bodies with which they are brought into relation, the latter are said to consonate with the former. Thus a note of another instrument, or of the voice, is sometimes reproduced by a guitar or pianoforte standing by.

[†] This is easily ascertained by running the gamut with the voice at the mouth of an empty water-bottle; one note only is intensified markedly, by consonance within its cavity—one or two others slightly. So, too, if a vibrating tuning-fork be held close to the embouchure of a flute, the note of the fork will be repeated by the flute, when the fingering belonging to that note is performed upon it; but with other fingering, the flute will be silent.

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* "The effect of carpeting or woollen cloth of any kind, in deadening the

Under the latter circumstances, bronchophony would, probably, (I have not verified this conjecture) be heard at some point of the chest nearer the bifurcation of the trachea; or, it is possible that certain changes of posture, altering the relationship of the reflecting surfaces, might interfere with the production of echo, by preventing the reflected sounds from coming to their usual focus; or, the position of the auscultator in respect of the focal point, might prevent him from hearing an echo really existing.* The force of the echo will also rise, the smoother the bronchial walls, and the larger the tubes in which it occurs; and numerous other circumstances may be conceived, but scarcely proved, to affect the phenomenon. Among these the composition of the gases within the bronchi (or in the pleura) may, for aught that is known, hold an important place: hydrogen has been proved to deaden sound greatly; the effect of carbonic acid, mixed with other gases and aqueous vapour, can only be learned by experiment.

Lastly, as concerns the distance from the point of auscultation at which the intensification within the thorax occurs:—the further away, the less of the resonance will reach the surface; the amount, however, will be modified by the conducting property of the interposed media.

A little consideration will show that these three conditions

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sound of music in an apartment, is well known. The intermixture of air and solid fibres in the carpets, through which the sound has to pass, deadens the echo between the ceiling and floor, by which the original sound is swelled."—Herschel, art. "Sound," Encyc. Metrop. Aerated mucus and sanguineous serum in the bronchi would have the same effect on vocal echo in those tubes as the carpeting under the circumstances referred to above.

* The theory of echoes generally is inadequate to explain many of their phenomena. There is (or was) a ruined fortress near Louvain well illustrating this. Here if a person sings, he only hears his own voice, without any repetition; those who stand at some distance hear the echo, but not the voice,—and they hear the echo with surprising variations,—sometimes louder, sometimes softer,—now near, now distant.—Burrowes's Cyclop., art. "Acoustics."

of bronchophony, conduction of laryngeal sound, intensification of this within the thorax, and proximity of site of the intensification, may or may not be directly as each other; one may be in a state favourable to, the rest unfavourable to, the formation of bronchophony. Hence the variable state of the sign in different cases of the same disease; and hence an easy clue, for example, to the inconstancy of bronchophony in pleuritic effusion.

There are two important varieties of vocal resonance within the chest (bronchophony), described by Laennec as special states of resonance, under the names of *pectoriloquy* and *ægophony*.

The essential character of pectoriloquy, as described by Laennec, is complete transmission of the voice through the stethoscope,—that is, a sensation as if the voice passed directly into the ear of the observer from the spot beneath. This condition of resonance he believed to be peculiar to, and a constant attendant on, excavations in the substance of the lung, except when accidental circumstances interfered with its production or propagation. As, however, he repeatedly met with excavations, when no such resonance had been discovered, he found himself constrained to admit "imperfect and doubtful" varieties of pectoriloquy—obviously nothing more than *common* bronchophony; but he does not seem to have been aware that most perfect pectoriloquy, as defined by himself, may occur where a *solid* mass, of medium size, is interposed between a main bronchus and the surface, and hence, under conditions, the most strongly opposed to those of excavation. The two kinds of fact show that Laennec's pectoriloquy gives no positive evidence of the presence of cavity in the lung; but they by no means disprove the reality of the peculiar propagation of resonance to the ear, described by Laennec.

We may then retain pectoriloquy as a variety of common bronchophony, under the title of *pectoriloquous*. It closely resembles the resonance heard over the larynx, and may exceed this in intensity; like the laryngeal voice, it appears to pass directly through the stethoscope into the observer's ear, and may throw the choncha, and even the neighbouring part of the skull, into more or less strong vibration. Limited generally to a small and accurately defined space, it may have a hollow and ringing character or not; though, generally speaking, loud, this is a wholly unessential property of pectoriloquous bronchophony, depending in great measure on the caliber of the laryngeal voice: the hollow and ringing characters, the insulation of the phenomenon, and its transmission, in an articulated form, through the stethoscope, may be distinctly marked, even when the ordinary voice is almost destroyed. When the physical conditions of its production (in a cavity of proper construction) exist in a patient thus reduced almost to a state of aphonia, it becomes peculiarly characteristic,—low muffled whispers pass directly into the ear, articulated sometimes with as much, if not more, precision than the laryngeal voice (whispering pectoriloquy): here there is no tactile fremitus on the surface, nor is any thrill communicated to the choncha of the listener,—proving that such thrill is not an essential element of pectoriloquous bronchophony. Even the loud pectoriloquy of a cavity may in each syllable be followed by a sort of whispering echo.

Bronchophony becomes pectoriloquous in certain conditions of excavation in the lungs, and in cases where solid masses lie between the bronchi and the parietes. If the quality of the resonance be markedly hollow and ringing, and if it exist in the *whispering* form, I believe, notwithstanding the statements made to the contrary, that it surely indicates a cavity: at least, I have met with no exceptional case; while, on the other hand, the most marked pectoriloquy of the *loud* form, without hollow and ringing character, I have almost ever heard, existed over a fibrous nodule in the pleura,—the lung being healthy and simply slightly condensed at the spot by pressure.

Whatever be the nature of the excavation, gangrenous, purulent, apoplectic, cancerous, or tuberculous (globular dilatation of a bronchus included), pectoriloquy *may* be perceptible. But it may not occur at all; or, if occurring, may be transitory or intermittent. The conditions of an excavation most conducive to its production are,—moderate size; smoothness and density of its internal surface, hence absence of bands either traversing its area or coasting its walls; emptiness; superficial position, and especially adhesion of its periphery to the parietes of the chest; thinness and hardness of that portion of its walls next the surface; and free communication with the bronchi. Where, on the contrary, a cavity is possessed of flaccid irregular walls, is more or less nearly filled with fluid, and deeply seated, with healthy lung interposed between it and the surface, the resonance will be wholly deficient in pectoriloquous character, and may be strongly or faintly bronchophonic, or *absolutely null.* Moderate size is of importance; small cavities (unless under special circumstances of seat) are rarely pectoriloquous; and very large dimensions are equally opposed to such resonance.* Very small diameter of the communicating bronchi impairs the distinctness of the phenomenon; and want of communication with the bronchi, also, will prevent its development, persistently or temporarily, according as the obstruction is itself permanent or dependent upon passing circumstances, such as accumulation of sputa in their interior. On the other hand, as observed by Laennec, where the number of fistulous openings by which a large excavation communicates with the bronchi increases, pectoriloquy becomes indistinct or ceases altogether; and if a communication be set up between a cavity and the pleura, or if the contents of the former escape into the subcutaneous cellular membrane, the phenomenon of pectoriloquy disappears. It follows very clearly from these facts, that pectoriloquous bronchophony must be frequently wanting in cases of caverns in the lungs, and that the other signs of destruction of pulmonary substance are much more trustworthy.

In regard of mechanism, pectoriloquy is either simple bronchophony in an intense form, or it is bronchophony echoed by the walls of a cavity.

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* Thus, (Green, U. C. H., Females, July 1850,) over an enormous tuberculous cavity furnishing perfect amphoric respiration with metallic echo, there is complete absence of vocal resonance of any kind.

When the vocal resonance has a metallic character, is not transmitted forcibly through the stethoscope, is not articulate, but conveys the impression of its being produced in a hollow space of large size, it is called *amphoric*, from the similarity of the phenomenon to that produced by speaking into an empty pitcher.

Ægophony (αιγος, gen. of *αιξ, a goat*, and *φωνή, voice*) the name given by Laennec to a special resonance, distinguished by its tremulous, nasal, and cracked character, suggestive of the bleating of a goat, is another variety of bronchophony. When most strongly marked, it is distinctly ringing, jarring, and muffled; is synchronous with the articulation of each word, or follows it immediately, like a feeble (sometimes whispered) echo of higher pitch than itself; conveys the idea of somewhat distant origin; does not appear to traverse the stethoscope, but rather to flutter tremulously about the applied end; is commonly persistent, but of short duration; audible over a very limited surface, and occasionally capable of being altered in position by varying the posture of the patient. Certain modifications of pure ægophony have been happily compared by Laennec to the voice passing through a metallic tube or cleft reed,—that of a person speaking with a counter between his lips and teeth,—the nasal twang of the exhibitors of Punch.

Though commonly persistent, ægophony sometimes loses intensity temporarily, reappearing after a fit of coughing or expectoration. Rarely lasting more than from two to five days, Laennec has known it continue in cases of chronic pleuritic effusion for several months. It does not accompany all notes of the voice, nor all words, even though pronounced with the same pitch: this peculiarity does not depend on the loudness of the laryngeal voice.

Pure ægophony is observed in certain cases, where a stratum of fluid contained in the pleura compresses the lung. The precise thickness of the layer of fluid most favourable to its production cannot be either laid down as matter of observation, or satisfactorily calculated. Laennec states that he has discovered this sign, when there were not more than three or four ounces of fluid in the chest. The ascertainable facts are as follow:—At the earliest period of pleuritic effusion, when it is deducible from physical principles that the fluid is tolerably equally spread over the pulmonary surface, there is commonly rather a tendency to ægophony than actual ægophony present. The lung is then—the inference arises—too slightly condensed, and the liquid accumulated too small in amount for the production of the phenomenon. It appears in fullest force during the period of gravitation, before any detrusion of the parietes has occurred, and consequently while the fluid is still moderate in quantity, and at its upper part spread thinly over the pulmonary surface. With the increase of effusion it disappears altogether, to return again (ægophonia redux) when absorption has reduced the liquid to a thin layer. So, too, in cases of paracentesis for empyema, ægophony appears after a certain portion of the fluid has escaped. On the other hand, exceptional instances occur (probably explicable by adhesions), in which ægophony remains in spite of very abundant accumulation. I have seen such cases, and such a one has been published by Andral (*Clin. Méd.*, t. ii., Obs. xxi.), where displacement of the diaphragm and heart gave evidence of the abundance of the fluid.

Supposing the patient to be examined in the sitting posture, the seat of the phenomenon will be found to be the neighbourhood of the inferior angle of either scapula (rarely of both), and a few inches on the side in a line with that angle; in very rare cases extending almost to the nipple in front. This limitation of seat is important as diagnostic of true ægophony. The shrill ægophonic quality of resonance, traceable to a naturally shrill and tremulous character of the voice, would be thus at once distinguished from resonance actually caused by the presence of fluid in the pleura, inasmuch as it would exist in the highest degree wherever the dulness under percussion was most marked; that is, commonly at the base of the lung. Now ægophony does not exist where the dulness is greatest; far from this: such a quantity of fluid as is capable of causing very notable dulness will almost inevitably (as just seen) cause the disappearance of ægophony, if it have previously existed. Exceptional cases are met with, however, in which the seat of true ægophony is more extensive. Laennec sometimes observed it over the entire affected side at the commencement of the disease. In two such cases he "ascertained, by examination after death, that this peculiarity depended upon the retention of the lung in partial apposition with the chest by means of pretty numerous adhesions, so that the lung became invested by a thin layer of fluid over its whole surface. In cases of this kind, the sign in question is observable during the whole period of the disease." But there is another cause why, at the commencement of pleurisy, ægophony (when it does exist) should be more generally diffused than at any later period of the disease. This cause, which escaped Laennec, because he mistook the true physical conditions regulating the mutual influences of the lung and fluid, is the equable distribution of the effused fluid in a thin sheet over the entire pulmonary surface—laminar effusion. Rarely, however, does the opportunity occur of observing pleurisy precisely at the period when the condition described exists; and actual ægophony is even then more unusual than a state of exaggerated resonance, partaking simply of the ægophonic character. It has been matter of inquiry whether ægophony is produced precisely on the level of the upper border of the pleural fluid, or at a certain elevation of that fluid where it has a certain thickness. The point is a difficult one to decide, but the conditions of the respiratory murmurs and of the percussion-sound appear, as a rule, to support the latter, the less commonly received, view.

Movableness of ægophony is essentially a character of short duration: the displacement of the fluid either ceases to be possible from the interference of plastic exudation, or the quantity of fluid increases to such a degree as to exclude altogether the conditions of its development.

Inflammatory and dropsical accumulations of fluid in the pleura are the states to which true ægophony is almost peculiar, —its existence in cases of hydropericardium is altogether exceptional; and the gravitating and absorption periods of pleurisy are those to which it particularly appertains. As double pleurisy is extremely rare, especially in persons free from tubercle or uræmia, while hydrothorax, on the contrary, frequently exists on both sides simultaneously, the absence or presence of ægophony on both sides would aid the observer in distinguishing inflammatory from passive effusion. In cases of pleuro-pneumonia, when fluid exists to the necessary amount in the pleura, the resonance of hepatisation becomes modified very usually by an ægophonic twang; but it is extremely rare to observe real ægophony in these cases. It is true that, many years ago (in the time of Laennec, indeed,) the apparently important discovery was made that ægophony exists in some instances of simple hepatisation; but the observation appears to me hardly a correct one. I have never yet detected ægophony of even tolerably pure character, as an attendant on simple inflammatory induration of the lung, unless the ordinary voice of the patient was of shrill tremulous character. Hence this exceptional species of resonance is most frequently encountered in persons, more especially women, of advanced age. I have been gratified by finding that M. Grisolle's experience (*Op. cit.* p. 242) has led him to a very similar conclusion. The bronchophony of hepatisation may, sometimes, be given an ægophonic character by directing the patient to speak with the nostrils closed.

Ægophony, according to Laennec, is the natural resonance of the voice in the bronchial tubes, rendered more distinct by the compression of the pulmonary texture, and by its transmission through a thin layer of fluid in a state of vibration. He thought it probable, also, that the flattening which the bronchi undergo from pressure of the pleural fluid had a good deal of influence in its production; the quality of resonance being such as might be anticipated from the shape of the vibrating tubes, resembling the mouth piece of the bassoon and hautbois: it is not sufficient in itself for the production of the phenomenon; otherwise, ægophony would exist in cases of absorption with *contraction* of the chest, which is not the case.* Laennec adduces various arguments in favour of these views, and affirms that by applying a bladder half filled with water over the larynx, the natural resonance is transmitted through the liquid with heightened pitch and slightly tremulous character. Skoda, holding that pure ægophony is audible in cases of pneumonia, and tuberculous infiltration, with or without cavities, as perfectly as where fluid exists in the pleura, and maintaining that a piece of liver interposed between the larynx and stethoscope will produce the same effect on the transmitted voice as a stratum of water, rejects Laennec's doctrine *in toto.* It appears "probable" to him, that ægophony arises only from impulses of a solid body against some other body, solid, fluid, or aeriform,—impulses which cannot occur within the chest, unless the voice consonates therein in a space filled with air; and that, "probably" in most cases, the wall of a bronchus, in which the air consonates, re-acts through impulses on that contained air, and so causes the tremulous sound; while it is also possible that mucus, &c., imperfectly closing the orifice of a bronchus, imitates the reed in the mouthpiece of reed-instruments, and so generates that sound. Dr. Sibson believes ægophony to be "pectoral resonance accompanied by whispering friction-sound; the two sounds are heard together, just as the drone and the notes of the bagpipe."

Dr. Sibson's theory seems inadmissible; because pure ægophony may be heard without the least shadow of friction-sound accompanying respiration, and because in cases of hepatisation with slight plastic exudation on the pleural surface, there may be abundant friction-sound, while the vocal resonance is totally deficient in ægophonic quality. Besides, the chest-motion during speech is scarcely sufficient to produce friction-sound,—which, at all events, could only be expiratory in rhythm. Dr. Skoda's idea of quivering mucus is likewise inadmissible; if correct, ægophony should be almost constant in a disease, in which no one has ever heard it, bronchitis: it should not exist in pleurisy with effusion, when there is no expectoration; and, wherever it exists, the respiratory murmurs ought to be ægophonic in quality, as well as the vocal resonance. As respects the same writer's notion of the quivering reaction of bronchial tubes on their consonating contained air, this would prove a great deal too much; for, if it were well-founded, the bronchophony of hepatisation ought *always* to be ægophonic: besides it leaves perfectly unexplained the occasional occurrence of ægophony in cases of fluid in the pericardium. For my own part, believing, as I do, that while other conditions may lead to a simulation of ægophony,* the pure quality described by Laennec never exists without the interposition of fluid, (it may be moved at will, in simple pleurisy, by moving the effusion,) I look for the explanation of the phenomenon in some degree at least to that fluid. Now Laennec's experiment is too rude an imitation of the state of things in the pleura to be trusted to, though a quivering character does very positively attend the resonance in the manner he affirms. The abruptness and the peculiar quality of ægophony are easily explicable by the intervention of liquid; the experiments of Colladon and Sturm have shown that the duration of sounds similarly produced differs notably in water and in air, and that their quality is completely different.

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* At least during the early period of such absorption; uniform or globular dilatation of the bronchi is well known to ensue at the advanced periods.

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* It seems not altogether philosophical to deny, with Skoda, the reality of ægophony as a special kind of resonance, because there are conditions of resonance which more or less closely simulate it; as well might the real existence of a special fluid, pus, be denied, because certain combinations of epithelium, salts and serum, are undistinguishable from it with the naked eye.

Thus a bell struck under water gives no tone as in air, but a quick sharp sound, as of two knife-blades clashed against each other.* Another fact discovered by these experimentalists, gives a clue to the rarity of ægophony: they have found that sonorous rays, which reach the surface of water at a very acute angle, do not pass into the air, but undergo a sort of reflexion in the interior of the liquid. Now the angle at which the sonorous vibrations reach the fluid from the bronchi, and ultimately reach the outer surface of the pleural fluid, may readily be conceived to be of the degree of acuteness fitted to prevent their passage to the air. The elevation of pitch in ægophony is inexplicable.

Before concluding the subject of vocal resonance, it may be observed that efforts have been made to connect peculiarities in the resonance of the observer's own voice (as he speaks with the ear applied to the chest directly, or with the intervention of the stethoscope,) with the amount of density of the parts beneath. Few auscultators can have failed to notice that while their voices resound with strong vibration from some chests, or from certain parts of these, no such resonance occurs from others: it is greatly more marked when the solid, than the hollow, stethoscope is used. I have, however, not found any condition of lung uniformly attended by, or uniformly free from, this sort of resonance (called autophonia by Hourmann); and consequently, in the present state of knowledge, attach to it no clinical value. I have known this resonance have an ægophonic quality in pleuritic effusion.

C. *The Resonance of the Cough* (*Tussive Resonance.*)—If the stethoscope be applied over the larynx or trachea of a healthy person while coughing, the act of expiration is found to be accompanied by a sound of hollow character, varying in respect of graveness and intensity with the voice of the individual; the observer is not conscious of any sensation of succussion in the site of its production. Auscultated on the surface of the chest, the cough in health furnishes a quick, short, commonly dull and indistinct, somewhat diffused sound, produced at a distance, without hollow or tubular character, not attended with a distinct sensation of succussion of the interior of the thorax.

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* Annales de Chimie et de Physique, t. xxxvi., pp. 243 and 254.

The modified states of the pulmonary cough, which occur in disease, are the *bronchial*, *cavernous*, *amphoric*.

Bronchial cough, when well marked, is a sound of harsh character; is attended with a sensation of very marked succussion in the chest, and a slight degree of impulsion towards the ear of the observer; is very rapidly evolved, and more concentrated under the instrument than the natural sound.

Cavernous cough is characterised by its perfect hollowness and metallic character. The sensation of production in an excavated space of limited size, the strong impulsion and transmission of the sound through the stethoscope with a force sometimes painful to the ear, are quite distinctive of this species of resonance. Cavernous cough may be pure, or associated with cavernous rhonchus; if fluid be present in the cavity to a moderate amount, it will not interfere with the production of the characteristic cough, and the forcible agitation the liquid matter undergoes during cough will of course be attended with rhonchus.

Amphoric cough is a loud resounding sound of metallic character, conveying the notion of production in a large space more or less empty; it is not forcibly transmitted through the stethoscope.

The varieties of thoracic cough are heard in the same cases as the corresponding varieties of respiration; they are of little utility in diagnosis. In pleuritic effusion, the quality of the cough is sometimes quasi-ægophonic.

D. *Phenomena common to the respiratory murmurs, to rhonchi, and to the resonance of the voice and cough.*—Differing from all the morbid conditions hitherto considered, the phenomena termed amphoric echo and metallic tinkling, attend the acts of respiration and of speaking. These phenomena are fundamentally one and the same,—the echo of various sounds, reflected by the walls of a capacious hollow space within the chest, under circumstances modifying the force, concentration, quality, and pitch of that echo. Metallic tinkling is the term originally applied by Laennec to a clear, ringing, highly metallic, single sound, of very high pitch, not dissimilar to that produced by gently striking a hollow glass vessel of globular form with a pin. Its quality may, however, be more or less purely metallic, and its pitch fall, and its clearness diminish (the tinkling character gradually disappearing *pari passu*), till it gradually merges in the low-pitched sound of buzzing amphoric echo. In different cases, or at different times in the same case, this transition may be detected; and there is a certain stage of the transition in which it is difficult to determine whether the term metallic tinkling or amphoric echo be the most applicable. The metallic quality, though less clearly and sharply defined, is yet very obvious in amphoric echo,—which may be imitated with some success by speaking, breathing, or singing into an empty water-bottle.

Metallic tinkling, occurring in connection with respiration, co-exists commonly with (or rather, echo-like, follows) inspiration, being prolonged somewhat into the expiration following, and is very rarely limited to the latter. Generally speaking, it alternates irregularly with an amphoric state of the respiratory murmurs, the one unnatural state giving place to the other, after a variable and for a variable number of respirations. It appears to be produced deep within the chest, or near the surface; and is rarely persistent for any considerable number of respirations. Amphoric echo may attend both sounds of respiration, or be limited to either.

Generally audible at the central height of the chest, laterally or posteriorly, (whence they may be propagated with gradually diminishing intensity to the surrounding parts,) metallic tinkling and amphoric echo may be heard in every part of the thorax. The most clearly marked and intensely developed metallic tinkling I ever heard, was chiefly audible under, and a little outside, the nipple: the case was one of tuberculous perforation of the pleura.

The mechanism of metallic tinkling and amphoric echo has been long sought after with all the eagerness of curiosity; but observers are far from having come to a uniform conclusion on the subject. I believe, as just mentioned, that the two phenomena are one and the same, fundamentally,—echoes of different properties from the walls of a large space, more or less favourably disposed for reflexion and concentration of sounds, produced either within the area, at the outlet, or in the close vicinity of that space. It appears, too, that the low-pitched buzzing echo only requires the presence of air in the hollow space, though water, in moderate proportional quantity, may be present therein; while the high-pitched tinkle requires fluid for its production, (not that such tinkle is physically impossible unless fluid be 'present, but that in the chest, the conditions, independent of fluid, which are capable of generating it, do not coexist.) In experimental support of this statement, it may be observed that if we blow, cough, speak, or sing, into an empty glass decanter, a low-pitched, buzzing, amphoric echo only will be produced; metallic and ringing in quality, it is true, but never of the tinkling pitch.* Let a little water now be placed in the decanter, and the result will be exactly the same, so long as the fluid is not agitated. But agitation of the fluid changes the quality of the echo. Thus, let drops of water, slowly, and at distinct intervals, fall on the surface of the fluid in the decanter, and the ear, applied to the surface of the vessel, recognises the most perfect imitation of metallic tinkling; just as in certain instances the phenomenon occurs within the chest, independently of respiration, rhonchus, voice, or cough, when a patient, with a very large cavity, or with hydro-pneumothorax, suddenly changes from the recumbent to the sitting or erect posture, and when, in all probability, a drop of fluid is precipitated from the roof of the cavity to the fluid on its floor. Or, again, breathe into the water by an elastic tube, and the bubbling will be found to produce a perfect tinkle.* It is probable, too, that sounds, generated in fluid, on the close confines of a cavity, itself free from fluid, may be echoed into metallic tinkle by that cavity. Metallic tinkle seems to be the echo of a bubble, or at least of a sound generated in liquid.

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* The fundamental and consonating note of such vessels is always of low pitch.

The morbid states in which these phenomena have been observed are hydro-pneumothorax with and without bronchial communication, simple pneumothorax, and large tuberculous excavations in the lung-substance. In the first case, (when the pleural cavity contains air and liquid, and opens into the lung,) both kinds of echo may occur with respiration, rhonchi, speech, and cough. If respiration produce in any way single, isolated bubble-sounds, (either by the bronchial fistula opening below the level of the liquid or otherwise,) metallic tinkling will occur; if there be no bubble-sound, amphoric echo only will be heard: hence, if the fistula open above the level of the liquid, there will be amphoric echo, unless the fluid be by some means or other simultaneously agitated. Rhonchi produced in the communicating bronchial tube or tubes, will be echoed into tinkle, if their component bubbles be separately, and, as it were, intermittently evolved; if otherwise, amphoric echo will be the result. The influence of speech or cough will similarly vary. Fournet has endeavoured to show that the occurrence of one or the other variety of metallic sound (tinkling or amphoric) will also be found to depend upon the freedom and rapidity with which the escape of air through the fistula occurs. If it make its way from the fistula by rare, slow, and successive bubbles, tinkling will be evolved; if the bubbles be numerous and closely following each other, amphoric echo will be the result. This idea seems to me well founded; if the drops of water be allowed to fall rapidly into the decanter (in the experiment I have already referred to), the sharp tinkle passes into the low-pitched and confused amphoric echo. Fournet further holds that (as the level of the fluid is in some cases capable of being changed with the position of the chest, and hence the relation of the fistulous opening to that level altered), the same opening may at one time be the possible source of metallic tinkling, at another of amphoric echo. Again, if the size of the opening increase much, amphoric echo will take the place of tinkling; and *vice versâ*, if its caliber be diminished by obstruction with pseudo-membrane or otherwise. Both phenomena will cease, he holds, if complete closure of the opening be effected. It is matter of certainty that both kinds of echo may occur independently of communication between the pleura and bronchi; this last statement is consequently incorrect. In the second case of non-fistulous hydro-pneumothorax, the agitation of the fluid by coughing or by movement, or the fall of drops of fluid from the upper to the lower parts of the pleural cavity, (perhaps even the echo of rhonchi in the adjacent bronchial tubes) will cause tinkling. Thirdly, metallic echo, and even tinkling, have been heard by some observers (the instances are singularly rare), to accompany both the voice and cough in cases of simple pneumothorax, in which there was neither liquid effusion nor perforation of the pleura. Low-pitched echo may intelligibly be produced under the circumstances; but the absence of fluid (especially where vocal sound is the alleged cause of the phenomena) makes it desirable that, in respect of tinkling, the observation should be repeated. On the rarity of simple pneumothorax it is needless to insist. Fourthly, large dimensions are an essential character of pulmonary cavities, that give either variety of metallic echo: all the four modes of production of both varieties may come into play within them.

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* The experiments of Dr. Bigelow (Bri. and For. Med. Rev., vol. vii. p. 569), made post-mortem on a patient with hydro-pneumo thorax, give similar results.

That the metallic phenomena should be (as is the fact) best heard in connection with coughing and speaking, is just what might have been anticipated: these acts require greater force of respiration than ordinary breathing; they are, therefore, on the one hand, capable of propelling air through a passage which would have resisted its progress under a less impulsion; and, on the other, they themselves being more sonorous, more readily lead to audible echo. Forcible and deep respiration will produce somewhat similar effects.

E. *Sounds of the Heart, and Murmurs, as transmitted through the Substance of the Lungs.*—In order to avoid repetition, I must refer the reader to the Second Chapter of this Part for an account of the mode and extent of propagation of the heart's sounds in the normal state of the thoracic organs: there, too, he will find the changes in transmission of its sounds produced by disease of the heart itself and of the great vessels considered. We have here only to do with cases where, the heart and great vessels being healthy, morbid states of the lung and its appendages (by changing the conducting power of the media intervening between that organ and the surface where auscultation is performed) pervert the natural mode of propagation. Now, inasmuch as the physical sources of sound in the heart are fluid and solid only, and aeriform matter has no direct connection with them, the displacement of air in the lung, either by fluid or by solid matter, would be favourable in theory to the conduction of the cardiac sounds through the pulmonary substance; while increase of air within the thorax would have the contrary effect. And so the positive intensity of sound produced in the heart remaining unaltered, its relative intensity, as discovered at different parts of the thoracic surface, might be changed.

Experience supporting, *in the main*, these theoretical considerations, teaches us that whenever the heart's sounds (the heart itself, great vessels, and chest-walls being sound) are found to be of greater intensity at any given point of the thorax, than at some other point nearer that organ, the lung, pleura, or mediastina have either in the *former* situation undergone some change, rendering them unusually good conductors of sound, or, in the *latter* situation, undergone some alteration diminishing their conducting power. The anatomical state in the first class of cases will be one of condensation or induration; in the second, of rarefaction.

Increased intensity of transmission of the heart's sounds is thus observable in pneumonia, chronic pulmonary consolidation, tuberculous disease, extensive pulmonary apoplexy, and œdema, dilatation of the bronchi, cancer of the lung, and solid accumulation in the pleura or mediastinum. The influence of pleuritic effusion will vary with its amount; the sounds will be better heard through a small extent of badly-conducting lung, than through a large mass of better-conducting fluid. Hitherto phthisis has been almost the only affection in which this means of diagnosis has been commonly applied. If the heart's sounds be more distinctly audible under the right than the left clavicle, and if the excess be sufficiently marked to leave no doubt as to its reality in the mind of the observer, the circumstance, in conjunction with the locality of its existence, affords presumptive evidence of tuberculisation. Generally speaking, other signs of a more direct character are observed at the same time; but in certain cases of incipient and rather deep-seated tuberculous deposition, it is often a source of satisfaction to have this additional sign to apply to. Its absence would not, however, by any means impugn positive evidence of consolidation derived from other sources. Whatever be the cause of the sign, it is for obvious reasons more readily substantiated at the right than the left side.

Diminished intensity of transmission of the heart's sounds, on the other hand, accompanies highly marked emphysema. The importance of this fact is habitually acknowledged in respect of the præcordial region, where the sounds may be almost completely muffled by an intervening thick mass of rarefied lung; but the sign may be established elsewhere, and, for obvious reasons, with greatest certainty on the left side. Thus, in a case of intense emphysema of the left lung (to which the disease was limited almost completely), and especially marked at the posterior aspect of the chest, I some years ago detected, much to my surprise, that the heart's sounds were considerably more distinct behind on the right than the left side. As there was no evidence of induration of the right lung, and as the sounds there were not louder than is sometimes observed in healthy individuals, the difference on the two sides could only be ascribed to diminished conducting power on the left. This sign, in the rare cases where it could be established, would appear to warrant the diagnosis of general emphysema of the substance of the lung in its deeper parts, as well as on its surface,—a point of some importance, for diagnosis (as well as anatomical investigation after death) generally aims too exclusively at the detection of *superficial* emphysema. The influence of pneumothorax varies; as a rule, it impairs the force of the sounds, but sometimes they seem to echo in the pleural cavity (as they do in a flatulent stomach), and so become intensified: I have observed this variation within twenty-four hours.

When the conducting influence is a weakening one, the observer should specially notice the second sound of the heart; when an intensifying one, the first.

The conduction of cardiac murmurs is modified on the same principle. But does any condition of lung generate either cardiac or vascular murmur, independently of disease of the heart, or of those conditions of the blood (spanæmic and other) which render its movement sonorous? It is by no means very uncommon, as Dr. Stokes was the first to state, to observe a sharp blowing murmur in the subclavian artery where the apex of the lung is consolidated by tubercle. Dr. Stokes ascribes the murmur to falling in of the infra-clavicular region, to consolidation of the lung, and to sympathetic irritation. He has found it remittent occasionally, and removed by leeching or by an attack of hæmoptysis; while it is completely wanting in the heart, aorta, carotid, or opposite subclavian. My observations agree almost completely with those of Dr. Stokes. But the *nature* of the consolidation is a matter of indifference; and I think this murmur is of rare occurrence, unless there be some systolic murmurishness at the aortic orifice. It is sometimes connected with a murmur at the second left (pulmonary) cartilage, evidently seated in the artery of that name; is greatly increased in force by suspension of the breath; sometimes disappears and reappears in the course of a few minutes; is sometimes removed by change from the sitting to the lying posture, and *vice versâ*, or even by brisk rotation of the arm. In quality the murmur varies from soft blowing to sharp whistling. It may continue for years, and seems more frequent in men than women. The pressure to which the vessel is submitted from the indurated and commonly *contracted* lung, especially when coupled with the least spanæmic tendency of the blood, seems its essential cause,—though the condition of the circulation in the part may impede or promote its formation.

Dr. Latham points out soft, blowing, systolic murmur, limited to the pulmonary artery, as a frequent concomitant of tubercles in the lungs. I have met with it occasionally, unassociated with subclavian murmur; but of this, more hereafter.

SECTION VI.—SUCCUSSION.

It has been seen that the succussion of certain contents of the chest, produced by the heart's impulse, and by the act of coughing, may give rise to physical phenomena of diagnostic import. And it was known to Hippocrates that if the chests of certain patients, labouring under thoracic diseases, be shaken, a "sound may be heard on the affected side." Hippocrates, however, erroneously supposed empyema to be the disease giving rise to this sound: his pathology was defective, but his observation correct: and the phenomenon retains to the present day the name of Hippocratic (or thoracic) succussion-sound.

The *succussion* necessary for the production and detection of this phenomenon may be performed by pushing the patient's trunk abruptly (but with gentleness) forwards and backwards, while the observer's ear is applied to the chest; or the patient may himself move his chest once or twice in the manner indicated. The *sound* resembles closely that perceived on shaking a decanter, partly filled with water, close to the ear. Like that, it is a guggling, splashing noise, the precise tone of which varies with the density of the fluid, and the proportional quantities of fluid and of air present. It differs in point of intensity according to the suddenness and force of succussion; but may be so easily produced as to be detected on the least movement of the patient, or during coughing. It may be audible at a distance from the chest, and be both heard and felt by the patient himself; and is, or is not, accompanied with metallic tinkling. Its duration varies greatly, —it may last for years, though this is very rare: in such chronic cases it is perceived by the patient, as he walks down stairs, rides on horseback, &c. It is not invariably a persistent condition when once developed; within twenty-four hours it may be present and cease to be producible, to recur again within a short period.

Produced by abrupt collision of air and liquid in an echoing space of large dimensions, the sound under consideration may be detected in hydro-pneumothorax, with or without bronchial fistula, and is occasionally to be heard in tuberculous excavations of unusually great size. Commonly audible over the general surface of the affected side, it may be limited to the anterior regions.* The sign is, however, by no means always to be discovered in hydro-pneumothorax: and one reason of its absence, thickness, and proportional excess of purulent fluid, was mentioned by Hippocrates †: it is certainly true that the thinner the liquid, the more readily is the sound produced.

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* Louis, Phthisie, éd. 2, p. 412. Paris, 1843.

† Laennec, by Forbes, Amer. edit. p. 541.

SECTION VII.—DETERMINATION OF THE SITUATION OF SURROUNDING PARTS AND ORGANS.

The object of attempting to determine the situation of other parts than the lungs themselves, when the diseases of these organs are the subject of investigation, is, as might be anticipated, to infer from any change in that situation the existence of some pulmonary affection capable of producing it. Experience has shown that such displacements, so produced, do occur: and further, that when present they are among the most conclusive (as they often are the most readily ascertained) signs of the pulmonary affection with which they are habitually associated.

The organs and parts liable to undergo displacement in consequence of pulmonary disease are—The *Heart*, the *Mediastinum*, the *Diaphragm*, the *Liver*, the *Spleen*, and the *Stomach*. The existence of displacement of these parts and organs is determined by means of various other methods of physical diagnosis,—by *inspection*, by *application of the hand*, by *percussion*, and by *auscultation;* very rarely by *mensuration*.

The *Heart* may be removed from its normal position by—(a) Detrusion, (b) Elevation, and (c) Procidentia.

(a) *Lateral detrusion*, for obvious reasons more readily detected when occurring towards the right side, is there commonly associated with slight procidentia; on the left, with some degree of detrusion, backwards and upwards. The progress of the displacement to the right side is usually gradual from its commencement till it has attained its greatest amount, when the organ pulsates between the fifth and seventh ribs to the right of the sternum. On the left it may be pushed almost under the axilla, its point being at the same time raised the width of an intercostal space, or thereabouts, and carried backwards towards the scapula.

Pleuritic effusion and hydro-pneumothorax are the affections which drive the heart sidewards to the maximum amount; simple pneumothorax is a rare cause; and hydrothorax, being generally double, does not displace the heart in this precise manner. Intra-thoracic tumors and aneurisms, variously placed, sometimes produce this effect; and hypertrophy, as well as emphysema, of either lung, are among its occasional causes. Besides, the heart may be *drawn*, as well as *pushed*, sidewards—a mode of displacement that occurs in some cases of rapid absorption of pleuritic effusion, of consolidation with marked contraction of the substance of either (but especially the right) lung, of pure atrophy (without distension of the air-cells), and also of great diminution of bulk from tuberculous disease [*] of the same organ. The practical interest of this matter is connected almost solely with pleurisy.

The heart can scarcely be pushed forwards by any lung-affection, except emphysema; and various more prominent conditions (such as the formation of a thick stratum of lung in front of the organ) tend to mask this displacement. Intra-thoracic tumors and aortic aneurisms lying behind the heart, push it forwards, and, especially in the latter disease, give rise to very peculiar signs.

(b) *Elevation* of the heart above its natural level, a displacement of very rare occurrence as a consequence of pulmonary disease (though sufficiently common in cases of abdominal tumor [†] and ascites), is sometimes seen as an effect of diminished bulk of the apex of the lung. Such diminution only occurs in tuberculous disease, and is produced by atrophy of the lung-substance, closure of air-cells, and contraction of exudation-matter, both interstitial and pleural. Dr. Stokes has known the organ pulsate under the second rib; I have never observed the apex higher than the fourth rib and third interspace.

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[*] I have twice, at the Consumption and University College Hospitals, seen the heart permanently beating in the right thorax, where no pleuritic effusion on either side had ever existed, as far as could be made out by present signs or past history, where the liver and spleen lay in their natural positions, and where great tuberculous excavation and destruction on the right side seemed to have *drawn* the heart in that direction, aided by perhaps the *detruding* influence of hypertrophy of the left lung.

[†] I have known the heart permanently raised an intercostal space by splenic enlargement. Case of Dujardin, U. C. H. Males, vol. v. p. 192. June, 1850.

(c) In *procidentia* of the heart the organ is below its natural level, and carried somewhat towards the median line; the impulse is then much more decided at the epigastrium (especially between the ensiform cartilage and left false ribs) than in the cardiac region. The common pulmonary cause of this displacement is double emphysema, of which it furnishes one of the most characteristic signs. It rarely exists to an appreciable extent in double bronchitis, if there be no emphysema; neither does double hydrothorax commonly induce it. The advance of tumors in certain situations may of course conceivably carry the heart downwards, but clinically this influence is rare.

The *Mediastinum*, at its lower part, is of course carried to the right or left by such morbid states of the lungs or pleura as produce lateral displacement of the heart. Superiorly, above the third rib, the mediastinum may be encroached upon by the lung (without any displacement of the heart), and sometimes pushed more or less to the opposite side. Emphysema of either upper lobe will produce this effect, and if both organs are implicated, the mediastinum may be, as it were, obliterated by its pleural borders being brought into close juxtaposition: the percussion-sound will of course be unnaturally clear. Tumor connected with the upper part of the lung, circumscribed empyema, tuberculous accumulation in the pleura, and acute hepatisation, may cause encroachment on the mediastinum, with dull percussion-sound: tuberculisation of the lung itself never produces this effect, the disease tending to diminish the bulk of the organ—hence occasionally a valuable aid in diagnosis. It is unnecessary almost to add that mediastinal tumors alter the relationships of the mediastinum; and that diseases of the great vessels, and of the heart likewise, deeply affect them.

The Diaphragm.—(*a*) In the *normal* state, the upper edge of the arch of the diaphragm, reaches, in the adult, the level of the fourth interspace on the right side, that of the fifth rib on the left, while the central tendon lies a little lower than this. In children the entire diaphragm rises somewhat less within the thorax. Full eating, and flatulent distension of the abdomen, temporarily raise it somewhat; and the habit of tight-lacing slightly depresses it.

The position of the right wing is ascertainable by percussion of the liver anteriorly; where the sound becomes clear on forcible percussion, carried from below upwards, lies the upper border of the liver, and (by inference) the convexity of the arch of the diaphragm. The cessation of vocal fremitus, where the liver is uncovered by lung, will corroborate the results of percussion, and supply a measure of the depth of liver covered by lung. The main guide to the position of the left wing will then be the fact that in health it always lies a little lower than its fellow; while the position of the heart's apex and the special resonance of the stomach will afford corroborative evidence. It has, besides, been shown by Edwin Harrison, that the exact situation of the vault of the diaphragm may, in many cases, be rapidly determined by inspection and application of the hand. The mode of proceeding varies according to the shape of the thorax, which is, with reference to this investigation, of two kinds:—1. If the width of the chest be greater just above, than precisely on, the level of a line drawn transversely across from the lower part of the ensiform cartilage,—in other words, if a slight lateral depression correspond pretty accurately to that level—a very simple method is described by this observer for discovering the position of the upper edge of the diaphragm. Let the hand be passed from below upwards along the side of the chest (its inner edge being kept closely to the surface and the palm somewhat everted), and that inner edge will sink into a narrow sulcus situated somewhat higher up than the lateral bulging just referred to. This sulcus, which may or may not be on the same level on both sides, indicates the precise height of, and corresponds to, the vault of the diaphragm. 2. If the width of the chest be less immediately above, than on the level of, the ensiform cartilage, this rule will not apply: however, the position of the left half of the septum may then be detected by the beat of the apex of the heart; and the right half is at least not lower than its fellow. In the main these guides are correct; but exceptions occur. Thus age, by enlarging the bulk of the lung through distended atrophy, or by diminishing that bulk through simple atrophy without distension, changes the position of the wings of the diaphragm, without affecting that of Harrison's sulcus. The sulcus is impressed on the side in youth, and remains unaltered in age. So, too, I have found that, where prolonged tight-lacing had driven the liver and diaphragm downwards, the sulcus had, for the same reason, ceased to correspond with the upper part of the arch.

(*b*) *In disease.*—The wings of the diaphragm may be both raised; both depressed; or one only may be depressed or raised, its fellow remaining *in statu quo;* or one may be raised and the other depressed; or the central tendon may be specially depressed.

In order to determine with precision the nature and amount of disease affecting these changes, I have been in the habit for some time of noting the position in the dead body of both wings of the diaphragm, before the chest is opened, and find in my hospital books thirty-three cases proper for analysis in this point of view. The highest position observed in these cases was the second intercostal space,—the lowest, three inches below the false ribs; the relative frequency with which the arches reached different heights of the thorax was as follows:*

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* It is to be remembered, that after death the collapse of the lungs draws the diaphragm slightly (but very slightly) upwards.

	Right.	Left.
Second space	1	1
Third rib .	–	–
Third space .	8	1
Fourth rib .	7	5
Fourth space .	8	4
Fifth rib .	6	13
Fifth space .	1	7
Sixth rib .	1	1
Below false ribs	1	1
	33	33

Hence it appears that in three-fourths of the cases the right vault lay *above* the fifth rib; while in two-thirds of the whole the left lay *opposite* or below it; and further, that the right wing in disease, most commonly lies between the third interspace and fifth rib (in $\frac{29}{33}$ of the cases); whereas the habitual range of the left wing is from the fourth rib to the fifth interspace (in $\frac{29}{33}$ of the cases).

Next, setting aside two cases of empyema, and one of ascites, I find that the right wing lay higher than the left in twenty-one persons; the left higher than the right in two; while both were on the same level in seven. Excluding the same three cases, the amounts of difference between the heights of the two wings varied thus:—

	Right wing, above left.	Left wing, above right.
By half a rib or space . . .	12	2
By a rib or space	5	
By a rib and a space . . .	8	
By two ribs and a space, or two spaces and a rib . . .	1	

Hence in nearly half the cases, where the right wing lies higher than the left, the excess of elevation only amounts to about half an inch.

But what are the morbid conditions connected with these variations in the position of the diaphragm? The case of elevation to the second interspace was one of enormous accumulation in the abdomen (ovarian and ascitic); both wings were equally raised; those of great depression (the diaphragm being highly convex downwards) were examples of great pleuritic accumulation (solid and fluid, and fluid and gaseous): these are the kinds of affection which seriously modify the position of the septum. A case where both arches lay opposite the sixth rib, was one of vesicular emphysema, uncomplicated with any other change of consequence. In the other cases a variety of conditions existed of opposing tendency; but a fair consideration of them leads to the conclusions—that rarefying diseases of the lung mechanically depress the diaphragm on one or both sides; that chronic condensing (because contracting) diseases raise it by a force of suction; that pleuritic adhesions, considered independently, have rather a depressing influence than otherwise: and lastly, that the discovery during life of any disease (even if it be highly marked) which tends *per se* to modify the position of the diaphragm, does not justify the assumption that it has so modified it in the particular case, for some counteracting (though less obvious) influence may be at play.

Now, it follows from the last proposition, that the position of the arches of the diaphragm in disease can only be ascertained by direct observation. Harrison's sulcus, remaining as it does a fixture, tends to deceive rather than enlighten; and (though the possibility of the change, in cases of old-standing disease, is conceivable,) I have never found a *new* sulcus corresponding to the altered site of the diaphragm. The vocal fremitus, the results of percussion and auscultation, are in truth the only real guides to the line of union of the chest and abdomen.

Extensive double emphysema, pericarditic effusion, and hypertrophy of the heart, lower the central tendon. The *liver*, *spleen*, and *stomach* may likewise be raised above, or depressed below, their natural level, by conditions altering the position of the diaphragm; and thus become affected with *Elevation*, or *Procidentia*. These alterations of position are more readily detected in the case of the liver than of the other organs named; and have for this reason attracted more attention on the right than left side.

CHAPTER II.

PHYSICAL EXAMINATION OF THE HEART AND GREAT VESSELS.

INTRODUCTION—CLINICAL TOPOGRAPHY OF THE HEART AND GREAT VESSELS.

THE heart, seated in the lower part of the anterior mediastinum, is held *in situ* by the great vessels (arterial and venous), and, through its pericardial covering, by the diaphragm. Lying obliquely, with its long axis directed forwards, downwards, and from right to left, the base of the organ corresponds, anteriorly, to the third, and the apex to the sixth, rib; posteriorly the base lies opposite the sixth and seventh dorsal vertebræ, separated from them by the aorta and œsophagus. The postero-inferior surface of the organ lies upon the central tendon of the diaphragm; the supero-anterior is in apposition partly with the lungs, partly with the walls of the chest.

Occupying the entire of the lower sternal, and certain portions of the left and right mammary, regions, the different parts of the organ are thus related to the surface. The right auricle reaches to the right of the sternum (more or less far, according to the state of the cardiac circulation) into the right mammary region, on the level of the third cartilage and interspace; while the right ventricle, mainly filling the lower sternal region, and coasting by its inferior, and nearly horizontal, border, the articulation of the sternum and ensiform cartilage, encroaches a little, at its base, on the right mammary region, about the fourth cartilage, and the two interspaces next above and below, and besides by its apex stretches into the left mammary region, at the fifth interspace.

The left auricle corresponds to the third left cartilage, and a

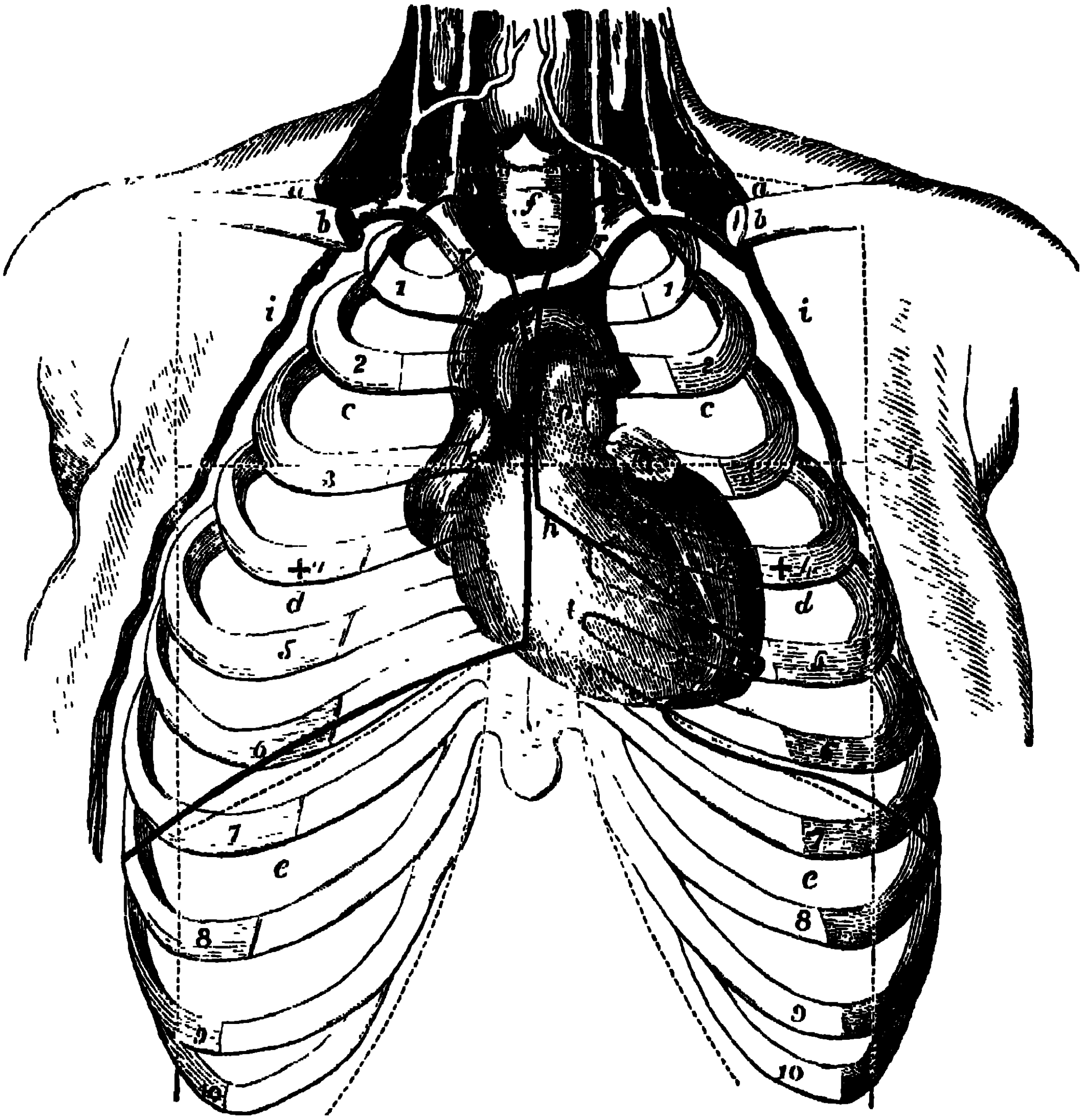

Diagram II., exhibiting the relationship of the heart and great vessels to the lungs (in moderate inspiration) and to the regions of the chest. 1 to 10 inclusive, ribs; *a*, supra-clavicular region; *b*, clavicular; *c*, infra-clavicular; *d*, mammary; *e*, infra-mammary; *f*, supra-sternal; *g*, upper sternal; *h*, lower sternal; *i*, integuments turned back; † †, nipples; *k*, right auricle; *l*, right ventricle; *m*, left auricle, appendix mainly seen; *n*, left ventricle; *o*, pulmonary artery; *p*, arch of aorta; *q*, vena cava superior; *r r*, innominate veins; *s*, innominate artery; *t t*, subclavian veins. The dotted lines indicate the outlines of the regions; the dark lines, the edges of the lungs. The heart and vessels are supposed to be full.

part of the interspace immediately below; the left ventricle to that portion of the left mammary region, lying a little within the vertical level of the nipple, and between the third and the fifth interspaces, or the upper border of the sixth rib. The heart, then, as a whole, extends, vertically, from the third cartilage to the sixth; transversely, from a little within the left nipple to about a finger's breadth to the right of the sternum: these are the limits of the *deep cardiac region.* The entire of the left ventricle, the greater part, by far, of the left auricle, and a fair portion of the right ventricle, towards the apex, lie to the left of the sternum; and on the level of the fourth cartilage, the widths of heart-substance, lying on either side of the left border of the sternum, are very closely the same. The most central part of the organ corresponds pretty closely to the sternal edge of the fourth left cartilage, or the upper angle of the fourth interspace. The organ corresponds posteriorly to that portion of the dorsal spine comprised between the fifth and eighth vertebræ.

The entire of the right auricle, and about one-third of the right ventricle, are covered by the right lung, which descends vertically downwards along the middle line to the lower edge of the heart; the entire of the left auricle, the upper left part of the right ventricle, and the entire of the left ventricle, except a variable, but small, portion towards the apex, are covered by the left lung. The portion of heart uncovered by lung, thus belonging almost exclusively to the right ventricle, is of rudely triangular shape,—the apex of the triangle corresponding to the middle line on the level of the fourth cartilage (where the anterior edge of the left lung diverges), the base to the lower edge of the heart, between the middle line and the spot at which the point of the heart beats: this is the *superficial cardiac region.*

The pulmonary valves are seated opposite the junction of the edge of the third left cartilage with the sternum; the aortic a very little lower and further inwards; the tricuspid and mitral valves lie at mid-sternum (the former in front of the latter), on the level of the third interspace. The transverse distance between the right pair of valves, averages half an inch; the left lie side by side; the aortic, quarter of an inch higher than the mitral:

an area of half an inch will include a *portion* of all four; an area of a quarter of an inch a portion of all except the tricuspid.

The aorta, rising opposite the confines of the two sternal regions, ascends at once into the upper, at first under cover of the pulmonary artery; and, inclining to the right, reaches the inner and upper part of the second right costal cartilage; thence crossing, almost horizontally, the upper sternal region, on the level of the first interspace, and in front of the trachea just above its bifurcation, it passes backwards to the left side of the body of the third dorsal vertebra. Sometimes the entire of the arch lies a little higher than this. The pulmonary artery ascends from the position of its valves, with slight inclination to the left side, as far as the second cartilage, its point of bifurcation; in this short course it passes somewhat backwards, and is consequently further somewhat from the surface of the chest opposite the second, than the third, cartilage. It occupies a portion of the upper sternal region, encroaching on the edge of the left infra-clavicular.

The arteria innominata rising on the level of the first interspace, behind the right half of the upper sternal region, passes upwards and to the right, bifurcating to the right of the trachea, and behind (or a little above, and to the right of) the sterno-clavicular joint: in its course it lies in front of the right half of the trachea.

The superior vena cava lies along the right edge of the ascending portion of the arch of the aorta, near the right border of the sternum, from the first interspace to the third cartilage; in the former spot occurs the union of the two innominate veins.

The pericardium reaches superiorly as far as the level of the second ribs.

The description now given applies with accuracy in the recumbent position only.

The action of the heart, besides altering its size, affects its position; during the systole the organ twists slightly on its longitudinal axis from left to right, especially towards the apex, which at the same time comes forward; the converse movements attend the diastole. But other modifications of position present themselves, independently of disease. In some persons the organ lies naturally a little higher or a little lower than the average, but any variation of this kind rarely exceeds the breadth of half a rib; the habit of tight-lacing in the female tends to depress the heart; the tight abdominal belt worn by some males to raise it slightly. Alteration of posture affects the site of the organ also,—the heart falls downwards somewhat (if its substance be weighty, the fall may equal an inch) in the erect position, and comes more forward than in decumbency; changing the position in decumbency from the right to the left side will carry the heart an inch, or even more, to the right or left of the position it occupies when the individual lies on the back.* Inspiration, by carrying the diaphragm downwards, lowers the heart, and, by bringing a thick stratum of the left lung in front of the organ, removes it somewhat from the thoracic walls; the weakening effect on the heart's impulse and sounds thus produced is in healthy persons very perceptible: the position of the valves and the maximum points of the heart's sounds are proportionably lowered; but it is to be remembered, that the depression of the diaphragm displaces the base more than the apex.

SECTION I.—INSPECTION.

In Health.—Inspection directs itself to (*a*) the form of the cardiac region, (*b*) the condition of its integuments, and (*c*) the visible impulse of the heart and great vessels.

(*a*) In perfectly sound chests the part of the walls lying to the left of the middle line, and corresponding to the heart, does not differ perceptibly in form from that placed to its right: these two divisions of the thorax are symmetrical. But individuals, who have never suffered from pulmonary or cardiac disease, occasionally present a moderate excess of convexity of the cardiac region, as a result of natural conformation, of curvature of the spine, or of change of form produced by influences of a non-morbid kind. So, too, physiological depression (or flattening) of the lower part of the præcordial region sometimes occurs,—but M. Woillez shows that such depression, when really non-morbid, is observed in the corresponding right region also,—is in fact symmetrical.

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* This is a point of considerable importance, as it shows that within certain limits, lateral movableness of the dull sound of the deep cardiac region cannot be accepted as evidence of fluid in the pericardium.

(*b*) The præcordial interspaces are of the same width, and lie on the same plane as their fellows on the opposite side; and the soft parts have the same characters on both.

(*c*) In the majority of healthy persons the heart's impulse is visible only at the apex, which beats in the fifth interspace, and somewhat against the sixth rib, about midway between the line of the nipple and the left border of the sternum; the area of its visible impulse does not exceed a square inch. Various physiological acts modify the precise spot of impulse. Thus variations of posture elevate, depress, throw it upwards or backwards: inspiration lowers it somewhat, and by carrying the lung in front of the heart weakens its force; expiration has the converse effects. A full meal or flatulent distension of the abdomen raises the apex-beat somewhat, and throws it to the left; pregnancy has the same effect. As a rule, thin, tall persons have an impulse of greater visible extent than the short and stout: in the obese, none can be detected; in persons with short sternum, it can be seen in the epigastrium. Habitually, it is more extensive in males than in females, and in persons of nervous than of other temperaments.

To the eye the impulse seems gently heaving and gliding, of brief duration, free from abruptness, and regular in rhythm.

In Disease.—(a) *Form.* The cardiac (or præcordial) region arches forwards in the course of pericardial effusion. I have never observed this change of form in pericarditis previous to the occurrence of liquid effusion; but it may occur while the fluid is yet very small in quantity. The intercostal spaces widen, and eventually almost protrude beyond the level of the ribs; while the left border of the sternum is pushed more or less (never more than slightly) forwards: change of form so marked as this indicates abundant effusion, and may reach from the sixth to the second left cartilages inclusive. Hypertrophy of the heart, especially of the left side, increases similarly the convexity of the cardiac region from the third to the seventh cartilages, and widens, but does not produce bulging of, the interspaces: pericardial adhesions, especially if associated with agglutination of the pericardium to the sternum, increase the bulging produced by any given amount of co-existent hypertrophy. Solid accumulations in the lower part of the anterior mediastium likewise arch the superjacent walls, and have occasionally caused great obscurity in diagnosis.

On the other hand, depression or excavation of the præcordial region may occur during the absorption-period of pericarditic effusion; commonly at the lower part of the region, occasionally, as I have once seen,* above its natural limits. In this instance the excavation formed during convalescence at the first and second left interspaces, close to the sternum. It has been the habit, on speculative grounds, to ascribe any such depression following pericarditis to the influence of adjacent pleurisy; but, in point of fact, the change of shape sometimes occurs where all physical signs have argued against the admission of the latter inflammation.

(b) *Integuments.*—The integuments of the cardiac region are sometimes markedly more œdematous than other parts of the chest in cases of pericardial effusion lapsing into the chronic state; the phenomenon, in itself unimportant, derives interest from some auscultation-signs, which may be traced to it;—these will be described hereafter.

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* Vide case of Craddock, Clin. Lect., p. 144.

(c) *Impulse.*—Disease alters the position, extent, force, character, and rhythm of the heart's impulse. The apex-beat is in the first place changed in position by a variety of diseases of the lungs, pleura, mediastinum, and abdominal organs* already referred to; but affections of the heart itself, and its external covering, likewise displace it. All enlargements of the heart depress the apex-beat, and may carry it as low as the seventh interspace, or eighth rib; if both sides of the organ be equally affected, that point is commonly displaced to the left; if the right side be the main sufferer, the impulse is chiefly visible to the right, behind and below the sternum; where the left side is alone, or chiefly, diseased, the apex may be seen at variable distances from the natural spot, to about three and a half inches to the left of the nipple.† If (as is not very uncommon) hypertrophy affect, almost exclusively, the part of the left ventricle adjoining the mitral orifice, the base of the organ falls disproportionately to the rest, and the apex is thrown forwards. Procidentia of the apex in general hypertrophy is, *cæteris paribus*, greatest where there is agglutination of the pericardial surfaces also. Aneurism of the arch of the aorta, or of the pulmonary artery, likewise lowers mechanically the point in question. It has been affirmed that atonic relaxation of the great vessels occurs in various acute adynamic diseases (especially continued fever) to sufficient amount to allow of an appreciable fall in the heart's level: I have lately observed an instance of this.

On the other hand, diminution of the size of the cavities, consequent on sudden enormous loss of blood, acting in conjunction with retraction of the great arterial trunks, slightly raises the apex-beat. It seems possible that a rude estimate of the degree of hæmorrhage might be formed through this changed position of the heart,—but of course only in individuals the precise point of whose normal beat was previously well known.

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* Enlargement of the right, as well as of the left, lobe of the liver, may displace the heart's point upwards and outwards, and sometimes give useful and unexpected aid in diagnosis. Such displacement contributed much to distinguish acute abscess with enlargement of the liver from abscess of the abdominal walls, in a case which I published some time since. (Case of Fairbanks, Clin. Lec., loc. cit.)

† The furthest point I have seen is 3⅝ inches outside the nipple, (*vide* case of Hope, Clin. Lect., p. 415); this, too, is only possible in broad-chested persons.

But the affection which most notably raises the apex-beat is pericardial effusion. When considerable accumulation exists in a pericardium free from old or recent adhesions, if the heart be not enlarged, and if there be no adjacent pleuritic adhesions, the increase of fluid pushes the base of the heart and the great vessels backwards and upwards, and twists the apex outwards and upwards, so that it beats opposite the fourth interspace or rib, behind (or a little outside) the nipple. The progress of elevation may be traced from day to day, as of the subsequent fall during the course of absorption. When the spot of the apex-beat is thus raised, it may always be seen, unless where general undulatory impulse tends to throw it into the shade; application of the hand will then either detect the point or not; if it fail to do so, the amount of effusion is in all probability very considerable. If during convalescence the apex gradually fall to below its natural site, hypertrophy, as an immediate sequela of the pericarditis, may be diagnosticated; for had the enlargement preceded the serous effusion, the apex would not have been raised.

In the natural state of things the maximum of the heart's visible impulse corresponds to the apex of the organ; under peculiar circumstances that maximum may be transferred to the base. Thus, in a case of highly developed hypertrophy and dilatation of the left ventricle,* where numerous circumstances pointed to the probable existence of an aneurism of the thoracic aorta behind the base of the heart, the impulse was very notably greater about the third than the sixth interspace. As the case did not terminate fatally in the hospital, I had no means of positively ascertaining whether aneurism existed or not; but I

* Case of Fredk. Groove, U. C. H., April 25, 1849.

do not believe that any affection of the heart's substance alone (except, perhaps, saccular aneurism of the left ventricle) will thus transfer the maximum amount of visible impulse from the apex to the base. I refer here to purely systolic impulse.

Instead of a forward movement accompanying the systole, a sinking inwards is sometimes to be seen. In some thin-chested people, with moderate hypertrophy, while the fifth interspace rises, the fourth sinks, with the systole; the organ appears to go through a see-saw movement, and produces a tendency to a vacuum at the fourth. So, too, the upper part of the epigastrium sinks in sometimes with the systole,—a sign originally deemed diagnostic of pericardial agglutination, and certainly, though more frequently absent, sometimes attending this state. I have known this systolic depression extend to the lower part of the sternum, and two or three adjacent cartilages, in hypertrophy with agglutinated pericardium. Hypertrophy with dilatation will produce epigastric depression during the systole, in persons with short sternums, independently of adhesions.

The other conditions of impulse, except the so-called undulatory variety, are better appreciated by the hand than by inspection. Undulatory impulse, suggesting the wavy motion of fluid, very variable in extent, may reach (in decumbency) vertically from the first to the sixth interspace, and transversely from the left nipple to an inch to the right of the sternum. Its position may be altered, in the same directions as the heart itself, by changing the patient's posture. Commonly the axis of undulation is diagonal, from below upwards, and from left to right: undulation may be well marked in one interspace, imperfect or null in an adjoining one; in this case the line of current is generally horizontal.

When so fully developed that there can be no mistake about the fact, undulatory impulse is a positive sign of fluid in the pericardium; but the sign unfortunately exists in a small proportion only of cases of hydropericarditis; and a kind of pseudo-undulation is not an uncommon character of the impulse of weak, dilated, and fatty hearts, especially where any physical conditions of the lungs or pleura, combining with its own enlargement, tend to bring an abnormal extent of the heart's surface in contact with the chest-walls.

SECTION II.—APPLICATION OF THE HAND.

By application of the hand, (*a*) the heart's impulse, (*b*) the movement of the præcordial ribs, and (*c*) the state of vocal fremitus over the cardiac region, are ascertained.

(*a*) The visible movement corresponding to the heart's point is found to possess a certain amount of impulsive force; it depends on the shock of the apex against the side. The essential clinical fact connected with this shock, is its synchronism with the systole of the ventricles; its mechanism is not thoroughly understood.

Though, of course, essentially the same in position as the visible impulse, the shock may be felt a little lower than seen, namely, behind the sixth rib. Slight in amount and imperceptible to the individual himself, mixed impulsive and gliding in character, free from abruptness or sharpness, yet decisive in rhythm, of brief duration, the force of the impulse is directly as the muscularity of the heart, and the energy and rapidity with which its fibres contract. However, in broad-chested and stout persons, though provided with powerful hearts, the impulse may be scarcely perceptible to the hand: it may then be detected by placing a stethoscope in the fifth interspace, and applying the ear as if for auscultation. In thin, tall persons, the impulse is disproportionably strong,—so also in women and children, in whom, too, it is distinguished by a certain abruptness and shortness.

Forced expiration widens the area and increases the apparent force of the impulse; inspiration affects both conversely. The varying condition of the neighbouring edges of the lungs fully explains this. Various acts quickening the heart's action intensify the shock,—muscular exertion, walking at a rapid pace, and, *à fortiori*, running, going against the wind, ascending heights, &c., have all this effect. Voluntary acceleration of the respiration will, especially in peculiar constitutions, act (indirectly, by quickening the circulation,) similarly on the impulse; so, too, will the slight erethism of the circulation that accompanies digestion. But of all causes which act in this manner, independently of disease, moral influences and the passions are the most powerful; and among these fear, perhaps, holds the highest place. The shock, under the agony of fear, becomes so powerful, as not only to be distinctly perceptible to the individual, but to be actually and sharply painful. Simple "nervousness" acts, to a slight degree similarly,—as the practice of medicine very constantly proves.

In adynamic diseases (*e. g.* continued fever) and in various non-febrile blood diseases, attended with depression of vital force (*e. g.* purpura), the heart's impulse habitually falls in strength.

Induration (acute or chronic) of the adjacent edges of the lungs, or abundant solid pseudo-membrane (induration-matter) in the contiguous pleura, solid substances in the mediastinum, and upward enlargement of the liver or spleen, lead to exaggeration of the heart's impulse; solid material being better fitted for conducting the heart's motion than spongy lung. Diminished size of the lungs, especially of the left, and pleuritic adhesions so placed as to withdraw the edges of the lungs from in front of the heart, or to cause by pressure atrophy of the enclosed parenchyma, intensify the palpable shock for obvious reasons. The falling in of the chest-wall, which ensues on these conditions of the lung and pleura, acts in the same direction. In all these cases, it will be observed, the exaggeration in force and extension of area are *surface-appearances*, and do not indicate any real increase of either on the part of the heart itself: many a supposed hypertrophy of the heart is nothing more than a simulation of the disease by some one of the physical conditions now referred to. On the other hand, emphysema and hypertrophy of the lungs, especially of the left, by bringing an unnatural amount of pulmonary tissue between the heart and parietes, weaken the palpable impulse, and may completely mask the direct parietal shock of well-grown cardiac hypertrophy.

Again, the quantity and quality of the circulating blood affect the heart's shock: at least the excited and sharply forcible impulse, occurring during reaction after hæmorrhage, appears in some part (perhaps frequency and force of propulsion in lieu of quantity propelled) due to the diminution of blood at command. In the spanæmia of chlorosis, the impulse, though sharp rather than strong, is yet stronger than in health. The condition of the heart's substance does not explain this.

Certain affections of the brain and spinal cord weaken the force of the heart's impulse; as do also certain medicinal substances,—aconite, digitalis, and hydrocyanic acid.

The diseased states of the heart which produce *real* increase in the force and area of impulse are,—morbid functional excitement (all the varieties of purely dynamic palpitation, angina pectoris, the paroxysm of "cardiac asthma," &c.), inflammation (cardiac and peri-, or endocardial), and enlargement, especially if combined with adhesions of the pericardium. The influence of inflammation is merely functional and dynamic; increased impulse only exists at the outset of pericarditis, before effusion has occurred to any extent. The influence of enlargement, on the other hand, is organic and statical; pure hypertrophy increases to its maximum the force of impulse; hypertrophy with dilatation, the force and area combined; dilatation weakens force, extends area.

The impulse may be increased to such a degree as to shake forcibly the head placed on the stethoscope, nay, even to shake the entire body of the patient, and the bed on which he lies; and it may stretch diagonally from the eighth left rib to the first right interspace close to the sternum, and transversely from two inches to the right of that bone to as many outside the left nipple: in such cases the impulse is very distinctly perceptible even in the left back. Between this, the maximum amount almost ever witnessed, and the natural state, all possible gradations are observable. Increased force, when extreme, is always combined with other modifications of impulse.

The force of impulse is lessened by effusions in the pericardium,—more readily in cases of passive than inflammatory accumulation, because in the former no excitement of the heart exists to intensify its action. Dilatation weakens the shock; fatty infiltration with softening has the same effect, although, on account of coexistent enlargement, the positive amount of impulse is above the average of health; abundant deposition of fat under the cardiac pericardium generally enfeebles the impulse.

Increased impulse may be especially perceptible either behind the lower part of the sternum from the fourth to the seventh left cartilage, and at the epigastrium,—or below and about the left nipple, and between this and the left costal cartilages; in the former case the right ventricle is commonly most affected, in the latter the left. But exceptions to this rule occasionally occur; extreme relative hypertrophy of the right ventricle will throw the impulse so much to the left of the sternum, as to simulate that of enlargement of the left side of the organ.*

If increased impulse be mainly traceable in a horizontal direction, the right ventricle is by some held to be the source of the increase, because enlargement on that side has a tendency to render the position of the heart more completely horizontal; whereas if mainly traceable in a vertical or vertico-diagonal direction, the left ventricle is at fault, inasmuch as its hypertrophy elongates the organ in those directions. I have occasionally found the former, frequently the latter, of these propositions correct; but neither is worthy of implicit clinical trust. Hypertrophy of the left ventricle, when extensive, widens the impulse horizontally.

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* Kernis, ætat. 10, U. C. H., Females, vol. ii. p. 237, 1847.

The character of the impulse varies as much as its force. The *apex-beat* may retain the natural impulsive and gliding character, and be merely increased in power. Or its impulse may acquire a quick, abrupt sharpness; or convey the sensation of slow heaving, or pushing forwards against an obstacle: in hypertrophy of the left ventricle, the latter character is habitual; in dilatation, the former. Where weakened, the loss of power may be simple; or the shock be also abrupt and jerking, or marked by fluttering unsteadiness, as in various species of softening. The *general impulse* on either side of the heart may vary similarly.

In rhythm, the heart's natural impulse is synchronous with the systole of the ventricles, and single. The impulse lags slightly behind the systolic sound of the heart, where fluid is accumulated in the pericardium, and the position of the apex is *not* elevated: before the impulse reaches the chest-wall, a stratum of fluid must be pushed aside. In certain states of disease it becomes double, the added impulse being systolic or diastolic. Double systolic impulse is an occasional, though rare, attendant on eccentric hypertrophy,—a not uncommon one (it may even be treble, whence a peculiar jogging, shaking character) where, in addition to the hypertrophy, the pericardial surfaces are agglutinated. Laennec noticed that in cases of great hypertrophy it seems "as though the heart swelled, and applied itself to the parietes at first by a single point, then by its whole surface, and finally sank back in a sudden manner." This sudden sinking back of the heart, Hope was the first to notice, is accompanied with a jog or shock (back-stroke) obviously diastolic in time. Hope believed it was occasioned by re-filling of the ventricles, and consequently more marked when their cavities were dilated, as well as their walls thickened, than in simple hypertrophy; simple dilatation he believed to be incapable of producing it. His ideas on its anatomical causes seem to me correct, but his explanation of the phenomenon may be doubted; and he does not sufficiently refer to the fact that the diastolic sensation is often rather one of *inward succussion* than *parietal impulsion*; it depends, quite as probably, on falling back of the weighty heart against the spine, —a view strengthened by the fact that "diastolic impulse" is peculiarly obvious in cases where solid accumulations (aneurismal or other) lie behind a hypertrophous heart. A *double* diastolic impulse has been described: this I have not observed,—it appears exceedingly probable that impulses, so described on the ground of their being unattended with arterial pulse, were in reality systolic, but too weak to reach the radial arteries.

In the normal state, any successive number of impulses are equidistant, and of precisely identical, or very nearly identical, force; in diseased conditions of the organ, very variable. There may be several strong shocks succeeded by others as feeble; and the number of each kind may be uniformly, or very nearly so, the same,—or their number may vary greatly,—in point of time all forms of irregularity are observable: force is rarely thus affected without time being implicated also. Simple hypertrophy of one or both ventricles does not produce this perversion of rhythm and force, unless some condition seriously affecting the pulmonary circulation occurs,—especially congestions, and inflammations of the lung. But both force and time are affected to a high degree, when dilated or simple hypertrophy of the left ventricle coexists with mitral insufficiency; force more than time, where the same state of the ventricle attends aortic insufficiency. Marked dilatation, fatty infiltration of the heart, and flabby softness, produce great irregularity of rhythm and force of impulse,—but no single impulse is, absolutely speaking, powerful, unless the walls of the ventricle be thickened also. Pericarditis, both before and after effusion, is occasionally attended with similar irregularity; so, too, are various malformations of the heart, especially during paroxysms of dyspnœa. In some of the latter cases perversion of the relative capacities of the two sides of the heart probably acts as the immediate cause of the irregularity.

The impulses, whether natural or morbid, hitherto spoken of, are caused by ventricular action: does the contraction of the auricles produce visible or palpable impulse? Certainly not, in the state of health: the tendency of their contraction is, indeed, to withdraw them from the surface. But I have recently seen a case of marked hypertrophy of the left auricle, where a strong impulse, slightly anterior in time to that of the ventricles, was felt in the third and somewhat in the second left interspaces close to the sternum.* It is probable, too, as suggested by Dr. Blackiston,† that the impulse of the ventricles may be communicated in some cases of dilatation of the appendix of the left auricle to the second left interspace: such impulse would, however, only be *pseudo-auricular*, and would be synchronous with the ventricular systole.

(*b*) The præcordial intercostal spaces are in the natural state of the same form and dimensions as on the other side of the chest. They are widened by hypertrophy, widened and even bulged outwards by pericardial effusion (especially in the young); narrowed by chronic pleurisy and pericarditis, especially if there be no hypertrophy sequential to the latter.

The state of motion in these spaces has been considered in the previous chapter.

(*c*) The normal effect of the heart on the vocal fremitus of the chest has already (p. 22) been explained. Enlargements of the organ (especially of its right division) and accumulations of fluid in the pericardium will annul the fremitus to an unnatural extent to the right of the sternum: but this is a sign of scarcely any value in practice, except where suspicion may be entertained that dulness under percussion, apparently connected with the heart, is in reality dependant on induration of the edge of the right lung.

Application of the hand detects, under special circumstances, two kinds of phenomena, which are purely adventitious—valvular thrill and pericardial friction-fremitus.

Valvular thrill (the purring tremor of Laennec) resembles, in many cases, the vibration of the surface of a purring cat,—in others is more like the vocal fremitus felt over the larynx of persons with powerful bass voices. Varying in force in different cases and in different conditions of disease, it may change from moment to moment in the same person, according to the energy of the circulation,—mental or bodily excitement rendering it powerful, when, in the state of repose, it had been almost imperceptible. Synchronous, and agreeing in rhythm, with the ventricular systole, it is felt mainly below and within the nipple, about the fourth interspace, over an area of from one to three inches, or at mid-sternum on the level of the third rib, in the third left, and the second right interspaces close to that bone: if perceptible higher than these limits, and especially if so above the clavicle, this species of thrill ceases to be of purely cardiac origin. Habitually caused by forcible and rapid propulsion, in a rippling current, of blood—more particularly of blood altered in certain of its properties—through orifices narrowed and roughened by disease, it may in rare cases occur independently of textural change in the valves. When connected with valvular disease, it ceases, for obvious reasons, to be produced, unless a certain quantity of blood is propelled with a certain force through the diseased orifice; and hence, after existing to a high degree, it may disappear, because the narrowing which originally led to it has increased to an extreme amount, while the heart's force has, from some independent cause, more or less failed; a similar alteration, it is well known, occurs previously to death in certain organic murmurs of the heart.

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* George Smith, U. C. H., Nov. 1850.

† Diseases of the Chest, p. 124.

The two combinations of disease, in which cardiac thrill is observed to the maximum degree, are insufficiency of the mitral valve with dilated hypertrophy of the left ventricle, and constriction of the aortic orifice, coupled with hypertrophy of the same kind and seat: in the former case, the seat of the phenomenon is below and within the nipple; in the latter, at the aortic base. Now, the physical conditions of the phenomenon seem pretty fairly balanced in point of completeness in these two combinations, yet certainly cardiac thrill is more frequently met with as a dependance on mitral regurgitant than aortic constrictive disease. Hope supposed that this depended on aortic thrill being masked by the interposed sternum; but, although it is true, thrill thus localised, may be sometimes rendered more distinct by causing the patient to lie on the left side, I believe the real cause of the comparative rarity to be no other than the less frequency of marked aortic than of mitral disease. I do not remember ever to have observed cardiac thrill synchronous with the ventricular diastole;* tricuspid and mitral constriction, as well as aortic regurgitation, have consequently not in my experience led to its production. The force of the current is, perhaps, insufficient in the three cases; yet, I can well conceive that in a spanæmic person, with highly developed aortic regurgitation, a minor degree of the phenomenon might exist. Neither have I ever known acute endocarditis (where there was full certainty of the absence of organic valvular disease) attended with thrill. But I have occasionally observed a faint aortic thrill (never a mitral one) accompany the systole at the base (when palpitation occurred from any accidental cause) in spanæmic women, and in persons who had suddenly lost a large quantity of blood. I do not believe that mere nervous excitement of the heart will produce thrill in people whose blood is healthy.

Pericardial tactile friction-fremitus, like the analogous phenomenon of pleural fremitus, is considerably rarer, and, when occurring, of shorter duration, than audible friction-sound. It has much more of a rubbing character than valvular thrill (with which it may co-exist), and is more moveable than this,—migrates from one part to another of the cardiac region within the course of a few hours, although the patient has retained and is examined in the same posture. In doubtful cases it may be distinguished from pleural fremitus by causing the patient to suspend his breath, for its rhythm is of course cardiac;* an excess of fremitus commonly attends the ventricular systole. Cardiac action may be supposed capable of producing pleural tactile fremitus; but I do not remember ever to have observed it. A remarkable case has been recorded by Dr. Swett (New York Journ. of Med., July 1840, p. 6.) where a distinct thrill over the heart was caused by friction against an enlarged and "tuberculated" left lobe of the liver; the heart (as likewise the pericardium) proved "perfectly normal in all its parts." Cardiac action may then produce tactile fremitus beyond the pericardium, and even through the diaphragm.

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* Skoda states that purring tremor "especially" attends mitral constriction; according to him the sign is, consequently, *diastolic* in rhythm.

SECTION III.—MENSURATION.

Mensuration of the surface corresponding to the heart, confirms inferences, otherwise deduced, as to the existence of certain affections of the organ; and sometimes becomes a useful auxiliary in diagnosis.

Thus, in health, the nipples are equidistant from the middle line; in enlargement of the heart and in pericardial effusion, the distance between the left nipple and that line undergoes increase, to a very notable amount in some old-standing cases of dilated hypertrophy. The left nipple, too, is carried somewhat downwards by the same affections. Again, the space comprised between the upper border of the third, and lower border of the sixth ribs, an inch outside the sternum, is the same on both sides in healthy persons free from curvature of the spine; the diseases just named increase the measurement on the left side. The influence of the two affections is the same on these measures; it differs in regard of the following one. The vertical distance between the left clavicle and the heart's apex-beat is in health the same as between the right clavicle and the point on the right side corresponding to that beat; in pericardial effusion, the measurement on the left side falls considerably below, in enlargement of the heart rises considerably above, that on the right side; this measurement is really useful in some cases of difficulty, as, for instance, when an enlarged and dilated feeble heart beats with a quasi-undulatory impulse.

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* Such difficulty must, however, be rare: pleuritic fremitus produces the sensation of successive *rubs*; pericardial, that of *thrill*.

In forced inspiration, the expansion on the level of the sixth cartilage, is in health slightly less on the left side than the right, a deficiency referrible to the influence of the heart. In pericardial effusion this deficiency, instead of (as might on first thought be supposed) increasing, actually disappears; the pressure of the fluid on the diaphragm throws an extra amount of work on the ribs, which move more comparatively than in health. When the fluid accumulates to a very great amount, however, say forty ounces, the præcordial ribs, also, move less than in health. I do not know how this matter stands previous to the occurrence of effusion in pericarditis; nor have I sufficiently examined the point to say positively how the costal motion varies in the different varieties of enlargement of the heart, with and without valvular disease.

SECTION IV.—PERCUSSION.

In Health.—Numerous difficulties stand in the way of accurate discrimination of the heart's outline by percussion: above the organ lie blood-vessels of large calibre, forming a quasi-solid mass; the lungs encroach on its edges; the liver, of a density and resonance scarcely differing from its own, coasts its lower border; while in front are placed the sternum, costal cartilages and ribs, possessed of a special resonance. Add to all this, that the heart is in a state of perpetual restlessness, and constantly changing its precise bulk and form, while the quantity of lung in front of it on either side is likewise undergoing the changes dependent on respiration. Yet, in spite of all this, it is possible by care to establish, with sufficient accuracy, for all practical purposes, the position and bulk of the organ.

The heart cannot be percussed with success except in the recumbent posture; and, unless for the special purpose of ascertaining to what extent the organ (or the source of præcordial dulness, whatever it be) is moveable, not the slightest movement, even of the limbs, ought to be permitted during the examination. The force used in striking will vary from the gentlest touch with the flat surface of the pulp of the middle finger, to a sharp tap with the ends of one or more fingers; it is a mistake to suppose that violent blows serve any useful purpose.

Percussion of the chest, where the heart lies immediately beneath the surface, gives a short, dull sound; the parietal resistance is highly marked. These characters are modified somewhat, except in the intercostal spaces, by the clearer and more prolonged resonance of the sternum, ribs, and cartilages. Where the organ is invested in front by lung, the resonance partakes of course, more or less, of pulmonary quality, according to the thickness of the intervening stratum of pulmonary tissue. We may then clinically speak of the heart's *superficial* and *deep-seated* dulness.

By reference to Diagram II., it will be seen that the limits of the heart's *superficial dulness* must be as follows:—on the right a vertical line, extending at mid-sternum from the level of the fourth rib to that of the sixth; on the left an oblique line passing outwards and downwards at a more or less acute angle from the latter, opposite the fourth cartilage, and curving inwards again (somewhat within the site of the nipple) to the sixth rib, beside the heart's apex; inferiorly, a line gently sloping to the left, from the central point of the lower edge of the sternum, along the sixth cartilage. This is the extent of heart uncovered by lung in calm respiration; and the form of the part is obviously, though only rudely, triangular. The vertical side of the triangle equals about two inches in length, in persons of middle stature; the horizontal about two-and-a-half inches, the oblique about three inches. This area is to be made out only by the gentlest taps with the pulp of a finger, either on the surface directly, or on a finger of the other hand. The right border is with difficulty established, on account of the sternal resonance. Forced inspiration diminishes the extent of this area; expiration increases it, especially in an upward direction and to the right; and by the kind of percussion described above, these changes in extent can without much difficulty be ascertained. They are, however, more frequently serviceable as tests of the freedom of play of the edges of the lungs, than of the condition of the heart.

Beyond the limits of superficial dulness, the outline of the heart may be ascertained by the deep-seated dulness elicited by full percussion; this *deep-seated dulness* extends normally in a vertical direction, from the third to the edge of the sixth cartilage, and transversely from the left nipple to a little beyond the right edge of the sternum opposite the fourth cartilage; the longest measurement is the diagonal one, from the upper part of the third right cartilage (the right auricle) to the point of the apex-beat. In ascertaining these limits there are two main sources of perplexity. The one consists in the difficulty of defining exactly the line of the base, as the dulness, produced by the mass of large vessels there, is nearly as great as that of the heart itself; this difficulty fortunately only occurs, where the edges of the lungs are unusually far apart. The other, the separation of the adjacent edges of the heart and liver, has already been discussed (p. 51). The "auscultatory percussion" of Drs. Cammann and Clark (*New York Journal of Medicine,* July 1840), most certainly renders the change of pitch, on passing from the liver to the heart, more positive and definite, than it appears under ordinary percussion, and may be called to our aid in puzzling cases. The method referred to consists in receiving percussion-sounds directly through a solid stethoscope to which the ear is applied, instead of, in the ordinary way, receiving them weakened by diffusion through the air. By practice the process becomes manageable without the assistance of a second person: the observer, having placed his ear to the stethoscope over the lower part of the liver, percusses the surface diagonally upwards and to the left, in the direction of the heart, and is apprised of his reaching that organ by an abrupt change in the pitch of the sound. The interesting paper of the American physicians is well worthy of study.

Certain physiological conditions modify the exact area of the heart's dulness. Its outline cannot be identical during systole and diastole; the chances are, that percussion gives the outline in a state of diastole or of quiescence: the chief interest of this matter turns, on the testing the correctness of results, obtained during life, by the actual size of the organ after death; it may be neglected in ordinary practice.* Again, alterations of posture of the trunk will of course change the position of the heart's dulness. The area of superficial dulness is relatively less in infancy, greater in old age, than in manhood.

(*a*) *In Disease.*—Various morbid conditions, independent of disease of the heart or its membranes, may increase the area of præcordial dulness. Among these may be enumerated *pure* atrophy of either lung, with *diminished volume;* consolidations of the portions of lung adjoining the heart; enlargement and elevation of the left side of the liver; accumulations in the mediastinum or pleura; diminution of bulk of the left lung with lateral pleural adhesions, a state tending to bring an undue quantity of the heart into contact with the chest-wall; aneurism of the great vessels; and even, in infinitely rare cases, tumor (with constriction and pouching) of the œsophagus. In all these cases the actual physical state of the heart may be completely unaffected. Not so, when the extent of præcordial dulness is increased in consequence of the stagnation of blood in the right cardiac cavities, caused by obstruction of the pulmonary circulation in dyspnœa, etc.: here the heart's own dimensions are temporarily changed.

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* The heart is drawn up after death, both by the emptying of its cavities and by the expiratory collapse of the lungs: hence another of several reasons why slight differences may arise in clinical and post-mortem examinations. The mode of death must seriously influence this.

There is only one thoracic affection independent of the heart itself which seriously diminishes the extent of præcordial dulness,—emphysema of the lungs,—especially of the left, and especially when conjoined with bronchitis. The temporary influence of bronchitis in increasing the bulk of an emphysematous lung, and so masking the dulness of a very greatly enlarged heart, is well shown in the case already referred to (case of Hope, Clin. Lect. loc. cit. p. 443). The same case illustrates the influence of ascites in diminishing the extent of the heart's dulness, by pushing the organ upwards under cover of the lung.

Venesection (as first shown by M. Piorry) will very sensibly diminish præcordial dulness, especially towards the right side, in persons whose right cavities had previously been loaded with blood. A marked state of anæmia, by reducing the heart's distension, narrows the area of its dulness.

(*b*) The area of præcordial dulness may be diminished or increased by disease of the heart itself. Diminution of that area attends primary concentric atrophy of the organ; but though decrease in bulk of the heart and in calibre of the large vessels, often occurs to a very notable amount in the tuberculous and cancerous cachexiæ, it is seldom detected during life in either: adjacent pulmonary dulness renders its discovery difficult in the former case; it is seldom sought for in the latter, but may sometimes be discovered, as in a case of carcinoma uteri, recently (January, 1851) observed at University College Hospital.

In pneumo-pericardium the natural dulness disappears more or less completely in proportion to the quantity of gas accumulated; even if there be fluid, as well as gas, in the serous sac, the entire præcordial region may give more or less tympanitic resonance in dorsal recumbency. By changing the patient's posture from the back to the side, dull sound will be elicited in the then inferior part, tympanitic sound in the then superior part of the præcordial region: this interesting fact I succeeded in establishing in a singular case of traumatic communication between the œsophagus and pericardium.*

The conditions which widen the area of præcordial dulness, are materially more important than those just reviewed; they are referrible to three main heads: enlargement of the heart, fluid accumulation, and solid accumulation.

(*a*) Hypertrophy, in all its observed forms and sites, increases the extent of cardiac dulness: the position of the increase and the elasticity of the walls differ under different circumstances. Simple hypertrophy of all the cavities, or of both ventricles, or the same state combined with uniform dilatation, extends the outline of dulness downwards, and to both sides, more, however, to the left than the right; scarcely any impression is produced on the upper outline of the heart's resonance,—a proposition which applies, with scarcely an exception, to all varieties of the cardiac enlargement; for, as already mentioned, the tendency of increase of bulk is to carry the heart downwards. In general dilatation, simple, or combined with attenuation, extension of dulness is also observed; probably very careful percussion might detect less parietal resistance in this than in the preceding cases. If enlargement be limited to either ventricle, the extension of the dulness takes place in the direction of the affected one; and what has been said concerning the site of impulse in such cases, applies to that of dulness. Accumulation of fat under the pericardium, when sufficiently great to alter the limits of dulness, generally does so to the right side; for the simple reason that such accumulation begins with, and attains its maximum at, that side. Hypertrophy of the left auricle carries deficiency of resonance into the second left interspace.

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* Case of Ramo Samee, U. C. H., under the late Dr. A. T. Thomson. The perforation was produced in the attempt to swallow a long blunt instrument, a juggler's "knife" (vid. p. 122); the case terminated fatally, and the preparation is in U. C. Museum. No. 3859.

If with the enlargement co-exist pericardial adhesions, the extent of dulness is always proportionately increased, but more so to the left than the right side: this effect is still more perceptible if there be pleuritic agglutination in the left inframammary region. In some cases of old pericarditis the area of the heart's dulness is increased upwards: thus I have repeatedly found, in persons who had previously been under my care for rheumatic pericarditis, that more or less marked dulness existed, years after, in the second left interspace, even up to the second cartilage, and at the adjacent part of the sternum. This state of resonance may exist with or without obvious enlargement of the heart; if without such enlargement, it can only be explained by the presence of solid induration-matter about the great vessels and base of the heart; if with such enlargement, it is explicable by the elevation, which the heart undergoes during the effusion-period of pericarditis, being maintained by agglutination of the pericardium, in spite of the depressing influence of the enlargement.

(*b*) Fluid accumulation in the heart's right cavities (of blood, of course) occurs to sufficient amount, under certain circumstances of obstructed cardiac circulation, to extend very perceptibly the area of dulness on the right of the sternum. The most important condition of the kind, practically, is dilatation of the right auricle and ventricle, combined with insufficiency of the tricuspid valve. The extension of dulness takes place mainly between the second and the fifth interspaces. The accumulation of fluid and semi-solid blood in endocarditis sometimes considerably widens the area, both to the right and left.

Fluid accumulation in the pericardium, whether passively or actively dropsical, or the result of pericarditis, produces an extension of cardiac dulness, even when of small amount. I know that four ounces will widen the area of dulness—perhaps even less than this will suffice. Clinical experience proves (artificial distension of the pericardium obviously could not be accepted as conclusive evidence) that the pericardium undergoes distension most readily upwards, with greatest difficulty downwards, with medium facility forwards, backwards, and sidewards. When distended with fluid, the sac retains its original pyramidal form—the base below, the apex above. The level of that base *at the front of the chest* commonly corresponds to the lower border of the sixth rib, sometimes to the sixth interspace, in very rare instances to the seventh rib: in the latter class of cases the texture of the pericardium had probably undergone some chronic change, of a rarefying kind, before the occurrence of effusion—or at least been affected with more than average facility by fluid pressure. In cases of extreme accumulation the diaphragm is arched downwards by the fluid; the epigastrium may thus be rendered somewhat prominent, but the dulness of the fluid is with difficulty (if at all) distinguishable from that of the liver,—it does not reach sufficiently to the left side to modify the percussion in the hypochondrium. The apex of the pyramid, as the fluid increases, gradually rises to the second left cartilage, to the first, to the sterno-clavicular joint, and even to nearly an inch above the clavicle,* displacing the apex of the lung at the inner aspect of the supra-clavicular region. As the fluid increases, it pushes aside the edges of the lungs, where they join in inspiration at mid-sternum, between the second and fourth ribs; * and this detrusion, in cases of abundant effusion, condenses the adjacent edges of the lungs, and so increases the lateral limits of dulness: on the right side the state of vocal fremitus will sometimes guide to the line where the condensed tissue and the fluid join. With equal superficial extent the dulness from pericardial fluid is more absolute, and the parietal resistance greater, than from hypertrophy;—this probably depends on the more perfect approximation of the fluid than of the solid material to the walls. This distinction is too delicate to be trusted to at the bed-side; the dulness of effusion is better distinguished from that of general hypertrophy by its extensive range above the third rib, and its limitation (commonly) to the sixth rib below; and from dilated hypertrophy of the right or left ventricle by not extending, disproportionately, to the area of dulness, towards the right or left side. It must not be forgotten that an aneurismal sac (we may suppose it filled with fibrine, pulseless, latent, and deceptive) of the transverse part of the arch of the aorta, and bulging inferiorly; or a small mediastinal tumor; or even superabundance of natural fat, placed just above the third left cartilage, and behind the sternum (a source of fallacy more frequent in persons with much subcutaneous fat than in the thin), may give to the dulness of an enlarged heart the pyramidal form of that dependent on effusion: and if an enlarged heart, with such an accidental appendage above it, be weak and flabby, and give a quasi-undulatory impulse, the diagnosis of the case (especially if its commemorative history be imperfect) becomes one of most serious difficulty.

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* Case of Bartlett, U. C. H. Male Cases, vol. iv. p. 292. Such extraordinary distension could only result from very slow progress of the disease: it appeared to have lasted here six weeks. The lungs were *five* inches apart on the level of the second rib.

(*c*) The plastic exudation-matter of pericarditis sometimes forms a layer, one-third to three-fourths of an inch thick, of solid substance applied to the heart's surface: if chance place this great thickness of substance on the lateral confines of the organ, the area of dulness must be proportionately increased; but the sign is one of too great delicacy to be clinically serviceable.

Cancerous accumulation in or underneath the pericardium affects the præcordial resonance in proportion to its amount.

The *quality* of the percussion-sound undergoes no very material change under any of the circumstances hitherto referred to; but in those very rare morbid states, induration of the heart's substance or ossification of the pericardium (Univ. Coll. Mus.), the sound must become more ringing and osteal in quality than natural,—at the same time the resistance of the parietes would be sharp and highly marked.

The *form* of deep-seated dulness (peculiarly affected, as we have seen, by pericardial effusion,) remains essentially unchanged by general hypertrophy; excessive hypertrophy of any particular part (for example, of the left auricle,) must of necessity modify the character of its outline,—but rarely to an amount that can be trusted to at the bed-side. Hypertrophy alters the form of the superficial dulness by pushing aside the lungs, and converts the triangular, into an irregularly square, space. Solid masses under the pericardium change the outline of dulness according to the direction of their growth; but their influence is very slight in this way, and on their rarity it is needless to insist.

Valvular diseases exercise no direct influence on præcordial resonance: the increase of dulness so frequently co-existing with them of course really depends on some form of attendant enlargement of the heart. The membranous inflammations, though probably slightly increasing the heart's bulk by congesting its substance, do not *per se* (endocarditis at any period, pericarditis until exudation has occurred,) appreciably alter præcordial dulness.

SECTION V.—AUSCULTATION.

Auscultation, directed to the heart, analyses:—(A.) Certain sounds produced by the normal action of the organ; (B.) Modifications of these occurring independently of heart-disease; (C.) Morbid states dependent on heart-disease; besides (D.) the state of the respiratory murmurs; and (E.) of vocal resonance, specially in the præcordial region, sometimes affords useful information.

In auscultating the heart, the patient should be placed in the recumbent posture, with the head slightly raised, unless, from the nature of his disease, this posture be an uncomfortable one.

As much of the precision of the notions, obtained from the examination, depends on our being able to connect the spots, where various sounds are heard, with certain parts of the heart itself or great vessels, it is obvious that, in order to simplify our task, we should *always* auscult patients in the posture in which clinical practice must present them most frequently to us.

If there be any doubt about the superiority of mediate or immediate auscultation, in the case of the lungs, there is none in that of the heart. Certain phenomena, well audible at a given point with the stethoscope, may cease to be perceptible *one-third of an inch* beyond that point: such limitation as this evidently could not be effected by direct application of the ear to the surface.

By forcible pressure of the stethoscope friction-sounds are increased in activity: when doubt is entertained about their existence, it may sometimes be thus removed. Sound produced by the heart's shock is similarly increased, and *pro tanto* the other elements of cardiac sound (or valvular murmur, if this be present) are thrown into the shade: hence, as sound produced by the heart's impulse often coexists with valvular murmurs, these are best heard through a stethoscope lightly applied.

The heart's sounds are more or less masked by certain morbid states of the respiratory sounds,—and they may be given unnatural characters by the chance coincidence of the sound of natural inspiration or expiration; hence the necessity of causing the patient to hold his breath from time to time, while the heart is ausculted.

(A.) *Normal Cardiac Sounds.*—Each complete revolution of the heart is accompanied by two successive sounds, audible in the præcordial region, and separated from each other by intervals of silence. These two sounds differ in all their characters; and the two periods of silence differ in the only character they can differ in, namely, duration: these differences are found (provided the position of the stethoscope be unchanged) to be maintained either without the smallest, or with very slight, variation in successive beats of the same heart.

The first of these two sounds, coincident with the systole of the ventricles, the heart's shock against the side, and the pulse (or diastole) of the arteries nearly adjoining the heart, is called the first, or systolic, or (because of maximum force at the lower part of the cardiac region) inferior, sound of the heart. The second of the two sounds, synchronous with the diastole of the ventricles, the recedence of the heart from the side, and the pulseless state (or systole) of the large arteries, is known as the second, diastolic, or (because of maximum loudness at the upper part of the cardiac region) superior, sound of the heart. The soundless period succeeding to the first sound may be called the first, or post-systolic silence; that succeeding the second sound, the second, or post-diastolic silence.

If the period of an entire revolution of the heart, that is from the commencement of one first sound to the commencement of the next succeeding first sound, be divided into ten equal parts, about four of these will be found to be occupied by the first sound, one by the post-systolic silence, two by the second sound, and three by the post-diastolic silence. This estimate is, however, only to be taken as approximatively true. When the pulse beats even as much as from seventy to eighty times in the minute, the post-systolic silence is difficult enough of detection; but it becomes obvious, where the pulse does not exceed sixty in a minute.

An accurate idea of the character of the heart's sounds cannot be given by a single description of them, as heard in any one particular spot: they vary materially at different parts of the cardiac region, both in their positive and relative properties. The least study of the healthy chest will convince the student that the description which it is the habit to apply to the sounds of the heart generally, holds good only when these are heard at the left apex. The sounds require comparative analysis: (*a*) at both sides of the apex-region, and at both sides of the base-region: (*b*) at base and apex on the same sides of the organ;

(*c*) at base and apex on opposite sides. Now this is the most difficult of all studies connected with the healthy heart; but it is essential as the basis of observation of the organ in a state of disease.

(*a*) At the left apex the first sound is dull, measured, booming, prolonged, and strongly accentuated; its commencement pretty sharply defined, its close much less so. Double the length of the second, deeply seated, and of lower pitch than this, it attains its maximum at this apex, in regard of accentuation, prolongation, and measured, booming character, but not habitually in amount of sonorousness. The second sound, only half as long as the first—clear, abrupt, flapping, and short—is more sonorous, more superficial, and of higher pitch than its predecessor. At the right apex-region, the first sound is considerably clearer, shorter, more abrupt, less strongly accentuated, and of somewhat higher pitch than at the left apex-region. This difference of character depends, probably, both on the thinness of the walls of the right, as compared with the left ventricle, and on the parietes being of different conducting power in the two situations: at all events the sound, audible at the end of the sternum, may be fairly referred more particularly to the right ventricle; that near the nipple, to the left ventricle. So, too, the second sound is habitually clearer, and sometimes even of higher pitch, at the end of the sternum, than towards the left apex; this is, however, less constantly true, and I have known the converse to be the case, where no suspicion existed of the existence of dilatation of the left ventricle—where, indeed, the first sound possessed to the full its natural share of dull, prolonged, booming character.

Passing from the apex to the base, the same kind of dissimilitude, laterally, in the sounds, may be heard,—not indeed precisely at the base, but a little above this, opposite the second interspace, where the aorta and pulmonary artery are placed side by side. The first sound at the right second interspace is commonly duller, of slightly lower pitch, and more prolonged than at the left corresponding point: in neither is it accentuated. The second sounds differ here in the same characters and in the same manner, but to a less degree: they are accentuated in both places, more on the right side than the left.

(*b*) Let us now examine the two sounds comparatively at base and apex on the same sides of the heart. The first sound, strongly accentuated at the left apex,—prolonged, booming, and dull,—at the left base loses the accent, which passes to the second sound, while this becomes louder, more ringing, and sometimes even of higher pitch than at that apex. At apex and base on the right side, the characters of the first sound are very similar; it has more accent in the former than in the latter spot, however, both positively, and *à fortiori* as compared with the second sound.

(*c*) The relative characters of the two sounds at base and apex at opposite sides of the heart (crucially taken, as it were,) may easily be deduced from the foregoing account.

Now, if these statements be correct, it appears obvious that no *single* articulate symbol can be devised, applicable to the heart's sounds in more than one point. The subjoined series is offered for the four points of prominent clinical interest. The acute accent-mark is used to show where the accent falls (twice, when it falls very strongly); the marks of long and short where one or other character is prominent.

	First sound.	Second sound.
At the left apex . . .	ū̋bb	dŭp
At the right apex . . .	úp	tŭp
At the left base* . .	up	tŭ́p
At the right base* . . .	ub	tŭ̋pp

The eye gathers from these symbols the tendency to equalisation in length of the sounds observable at the bases, as well as the transference of the accent from the first to the second sound at the apices and bases respectively.

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* The left and right second interspaces close to the edge of the sternum.

The extent to which the heart's sounds are audible in health, is not subject to any fixed rule. One great mistake,* commonly committed by authors who attempt to define it, is not considering separately the first and second sounds. From this omission, the ordinary starting proposition, that "the heart's sounds are heard at their maximum in the præcordial region," becomes an error: the second sound is, in truth, heard in nine people out of ten, more clearly at mid-sternum, on the level of the second interspace, than at any point of the præcordial region,—even limiting that region to the space in which the heart is uncovered by lung during tranquil breathing. The thickness of the soft parts, the form of the chest, and many other physical conditions, perfectly independent of disease of any of the thoracic organs, modify the extent of propagation so variously, that there can be no practical utility in laying down rules subject to perpetual exceptions. But the lines of propagation of the two sounds severally agree in most healthy persons, whatever be their absolute intensity at their seat of production; changes in these lines point positively to some modifying cause, and hence their establishment is clinically valuable. Now, the first sound passes slantingly upwards to the left acromial angle, growing weaker and weaker on the way; it loses much more on the way to, and at, the right acromial angle: its propagation backwards is clearest and fullest to the left,—so that while audible at the left back, it may be inaudible at the right. The second sound, with the base-region as its centre, radiates to the right and left acromial angles, with greater clearness to the left than the right; posteriorly it reaches the surface at the right side less clearly than at the left.

The difficulty of unravelling the mechanism of the healthy sounds of the heart is emphatically proved by the fact that, from the time of Laennec to the present day, at least twenty-nine theories have been proposed in its explanation. It would be a tiresome, and indeed useless, task to review these theories *seriatim:* the better plan seems to be to state with as much brevity as possible the rationale of the sounds, supported by the greatest amount of clinical and experimental evidence.

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* Some authors speak of the sounds being audible in healthy male adults, of moderate stoutness, even *at the right side posteriorly;* others hold that the space over which they are heard *seldom exceeds two square inches!*

The occurrences which are possible sources of sound, and with which the first sound is coincident, appear to be,—at its commencement, the impulsion of the blood in the ventricles against the auricular valves; the sudden tension of these, and the sharp collision of a portion of their surfaces; the attrition of the blood-elements *inter se* within the ventricles, or their impulsion against their walls; the projection of blood from the ventricles against the orifices of the pulmonary artery and aorta, and the columns of blood contained within them; the shock of the heart's apex against the side, or against lung-substance, if this be interposed; and the attrition of the pericardial surfaces near the apex;—towards its close, when their contained blood has been expelled, the collision of the surfaces of the ventricles;—and, throughout its entire duration, the sustained muscular contraction of the walls of the ventricles, with, at a period varying with the condition of the heart's substance, the perfect tension of the muscular fibres. Now, if we except pericardial attrition (inasmuch as the healthy first sound has no shade of friction-quality in it*), and, probably, collision of the blood-elements among themselves within the ventricles (inasmuch as experiments seem to show that such collision is, at the least, rarely sonorous), all these phenomena are more or less constant, and more or less powerful causes of the sound. That the sound derives its dull, booming prolongation from muscular contraction, seems unquestionable, not only because it retains these characters when the heart contracts after separation from the body, and the action of the auricular valves is prevented, but because it may in these characters be pretty closely imitated by the contraction of voluntary muscles.* Nor can there be any doubt that the tension and surface-collision of the auricular valves, and sharp shock of the blood against their ventricular surfaces, give the comparative sharpness to the first sound,—a character which may be detected by attention at its outset, and which, in certain states of altered contractility of the muscular fibres, almost covers or rivals in strength the sound generated by these: the elements of sonorousness exist in these conditions; and, when they are experimentally interfered with, a corresponding change follows in the character of the first sound. The projection of the ventricular blood against the orifices of the large vessels, the flattened valves, and the bases of the columns of blood they contain, combined with the sudden extension of the arterial coats beyond, have strong clinical and experimental claims to a share in the first sound. A sound is audible in the arteries synchronous with the heart's systole, under circumstances in which the idea of mere conduction from the heart is quite inadmissible: such sound may be heard in the femoral and even popliteal arteries sometimes, where no disease of these vessels or of the aorta exists. Again, in certain cases of mitral regurgitant disease, where the systolic sound at the left apex is completely deficient (a murmur only existing there), the first sound may be discovered with the quality of health at the aortic base. There is a want of clear evidence that the collision of the surfaces of the ventricles contributes to the production of the sound at its close; but it seems, at the least, probable that such is the fact,—and that the clicking character of the sound, after profuse hæmorrhage, may, in part, be due to this cause. Lastly, the heart's shock against the side, especially when the posture of the individual, the period of the respiratory act, or other conditions, allow the surface of the organ to play fully against the parietes, indubitably increases the first sound, and gives it in particular cases a *knocking* character.*

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* This is, in my mind, a much more conclusive reason for its rejection than the fact that the first sound continued, not *obviously* changed, after the pericardium had been removed in living animals. Such continuance would merely prove that pericardial attrition took no *prominent* part in generating the sound. But in exceptional cases (and where there is no reason to believe the pericardium diseased), the first sound has a distinct, though slightly-marked, attrition quality. This was evident, for instance, in a woman (Roberts, U. C. H., Oct. 10, 1850,) with general moderate dilated hypertrophy, in whom the knock of the heart against the side was *occasionally* distinctly sonorous, and accompanied with a sensation of faint rubbing palpable to the hand: all three signs disappeared under rest and appropriate treatment.

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* In a remarkable case (which I saw with Mr. Hardwicke) of hypertrophy of the recti abdominis, with contractions (partly reflex, partly voluntary,) of their tissue, the variation in intensity of the sound, according as the contractions were slow or quick and abrupt, was very remarkable: in the latter case, the sound had a very distinct resemblance to the first sound of the heart.

The essential causes of the first sound, then, seem to be muscular action (walls of ventricles), valvular tension (auricular valves), and forcible shock of fluids against resisting membranes (orifices of large vessels); while various subsidiary causes act occasionally, especially impulsion of solids against solids (heart's apex against the chest walls).

The phenomena synchronous with the second sound are: the diastole of the ventricles and rush of blood into their cavities; the sudden recedence of the heart's apex from the chest walls; the abrupt fall of the auriculo-ventricular valves to the sides of the ventricles; the sudden tension of the sygmoid valves, and impulsive fall of the columns of blood against them during the arterial systole; the arterial systole itself.

Now, of all these causes, the most effectual is (as originally taught from clinical observation by Dr. Carswell†) the tension of the sygmoid valves: the absolute disappearance of the natural second sound at the aortic orifice, and its persistence at the pulmonary orifice, in cases of insufficiency of the aortic valves, is a sufficient proof of the fact. The quality of the sound, and the site of its maximum force, as already described, depose, too, in favour of its membranous origin, and of its localisation at the orifices of the great vessels.* The fall of the columns of blood on the surface of the valves (though not so sonorous as if the valves, instead of being opened out by the receding blood, as they are, were *first* expanded, and *then* received the shock of the fluid from above, as is affirmed to be actually the case by M. Hamernik,) must intensify the sound of valvular tension. Whether the arterial systole itself plays any part in its production, is yet open to inquiry.

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* Where a thick layer of emphysematous lung intervenes, and sometimes even without this, I think there is reason to believe the first sound may be given an *intermittently murmurish character* at the apex, independently of any disease in the mitral valve, simply by the apex-point of the heart impinging against the lung, and moving some of its contained air. I have observed the phenomenon (during suspension of the respiration) where this seemed its sole plausible explanation.

† See Archives gén. de Méd., t. xxvi., 1831.

Whatever be the force of the arguments in favour of the *active* character of the diastole of the ventricles, it seems certain, as matter of experiment, that the phenomenon is soundless. Skoda argues, that as exceptional cases occur in which the second sound is weak at the base, and loud and clear at the apex (while there is no diastolic impulse against the chest wall), it must originate in part in the ventricular region; and suggests that it may be produced either, "perhaps, sometimes," by the stroke of the blood against the ventricular walls during the diastole of the heart; or by separation of the heart's apex from the surface, against which it had been pressed during the systole; or by separation from the parietes of the portion of the pericardium which had been driven against these during the systole. I am disposed to believe that there may be more truth in this notion than there seems on surface-consideration of it: if the chest be percussed with the point of a finger, while the ear is applied to a solid stethoscope in a neighbouring spot, two sounds are heard for each blow; the first strong, corresponding, of course, to the direct impulse; the second, very weak, to the removal of the finger from the point percussed. Now the recedence of the heart's point from the side is here imitated. Further, in the normal state, the blood enters the ventricles from the auricles, with a current so calm as to render it singularly unlikely that audible sound can be the result. But in cases of highly marked aortic regurgitation, blood falls with notable force into the left ventricle, and may conceivably generate sound. I have unquestionably heard, at the left apex, a distinct sound in more than one such case, while at the aortic base the ordinary regurgitant murmur alone existed: such cases would probably be more frequently met with, were it not for the loudness, and easy transmission to the apex, of the murmur at the base. I am persuaded this intensified second sound cannot be the transmitted sound of the pulmonary valves, because I have found it stronger at the left than the right apex.*

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* The experiments made on large animals leave the mechanism of the second (and, *à fortiori*, that of the first) sound far from satisfactorily established: the matter really rests on clinical evidence. Thus, in Hope's records of experiments in which both pulmonary and aortic valves were hooked up, the simple statement is made, that the "natural second sound entirely ceased, and was replaced by a prolonged hissing." (Dis. of Heart, Ed. 3, p. 35) We are left in the dark as to whether the second sound was thus ascertained to have become inaudible at the base only of the heart, or over its entire surface. From several passages in the context, the former seems the more likely; and hence the records at least of these experiments leave it still an open question, whether or not the natural second sound is in any small degree ventricular in site, or capable of becoming so, when aortic regurgitation exists.

M. Magendie's idea of a *normal* diastolic shock of the heart against the side is illusory; and the fall of the auricular valves against the ventricular surface is not demonstrably sonorous. Hence the sudden tension of the sygmoid valves, and the fall of blood upon them, is in the *normal* state the sole demonstrated cause of the second sound.

(B.) *Modifications of heart's sounds occurring independently of cardiac disease.*—The description now given of the sounds of the heart applies especially to the male adult. The first sound is clearer and more ringing in females, hence apparently more intense, and, *cæteris paribus*, more extensively audible. The same statements apply to children.

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* In a certain proportion of cases of mitral regurgitation and dilated hypertrophy, an intensified second sound may be found at the left apex;—its explanation will be sought hereafter.

Variation of posture has little, if any, effect on the second sound; but the first is commonly stronger in the erect than in the reclining, and in the prone than in the supine, postures; the reasons are obvious.

The extent and direction of transmission of the healthy heart's sounds are modified by various changes in the conducting power of the contents of the thorax (p. 146); unless the absence of such changes has been established, we are not justified in inferring that a plus or minus extent of propagation depends on the state of the heart itself. Enlargement upwards and inwards of the liver or spleen renders the sounds unduly audible in the hypochondria.

When the heart is displaced by diseases extraneous to itself, it might be supposed that the points of maximum force of its sounds would likewise be displaced: this is true, however, to a much greater extent of the first sound than of the second, and for the evident reason that the apex of the organ is more moveable than its base. In left pleuritic effusion, for example, when the maximum point of the first sound is carried to near the right nipple (say seven or eight inches out of its place), the maximum of the second scarcely swerves farther than to the right edge of the sternum, or one and a half inches from its post. Now it follows from this, that the line connecting the maxima points of the two sounds deviates, more or less, from the almost vertical bearing it presents in health.

The condition of the hollow organs of the abdomen, especially the stomach, sometimes curiously modifies the quality of the heart-sounds. When distended with gas to a certain amount, the sounds echo within it with a metallic ring, and so loudly, sometimes, as to be perceptible to, and seriously alarm, the patient.* Large cavities in the adjoining lung (especially if their walls be hard), and the pleural sac distended with gas, sometimes echo the heart's sounds in a similar way.

The intensity of the heart's sounds (and even the pitch of the first) is heightened by nervous excitement of all kinds: in the hysterical and epileptic paroxysm this is sometimes strikingly remarkable; emotion, whether of fear, anger, &c., has a similar effect, the sounds being audible even to the individual himself, and to by-standers at one or two feet distance from the chest. Diseases of debility weaken both sounds; but this effect is greatly more perceptible in the first than the second: the causes of the first sound give the clue to this difference.

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* Case of Warren, U. C. H., Oct. 1849. Aneurism of arch of aorta.

Certain conditions change the combined force and quality of the first sound by influencing some one of its elements without affecting the others. In continued fever, the general weakness, impairing the muscular power, throws the valvular element into undue prominence, and gives the first sound a clicking character, akin to that of the healthy second. Again, nervous excitement intensifies and gives a ringing quality to the impulsive portion of the sound, so much as to throw the others into the shade. The second sound is comparatively little affected in either case.

The number of sounds corresponding to each revolution of the heart may be increased (but not diminished), independently of disease in itself, in the manners to be presently described.

(C.) *Morbid states dependent on heart-disease.*—These are referrible to the heads of—I. Modified sounds; and, II. Adventitious sounds or murmurs.

I. *Modified sounds.*—The intensity or loudness, and the extent of transmission of the sounds, when modified by heart-disease, are in the direct ratio of each other. *Increase* in both respects is observed to its maximum in hypertrophy (especially of the left ventricle), with considerable dilatation, the valves remaining sound; muscular substance exists in sufficient excess to intensify the muscular share of the first sound, while the thinness of the walls, as compared with the size of the whole organ and capacity of its cavities, acts as a special source of loudness. The forcible propulsion of the blood into the aorta leads to proportionally forcible reaction of the arterial coats, and unusually sharp recoil of the column of blood in the sygmoid valves. In simple dilatation, the first sound, louder than in health, is sharper and clearer also: the heart is brought more extensively than natural into contact with the chest walls, and the thinness of the muscular walls allows of short abrupt contraction. Induration of the walls of the heart weakens the muscular portion of the first sound, but intensifies the valvular; and (if the other conditions for this exist) gives peculiar loudness to the impulsive noise produced by the apex.

Functional excitement of the organ also intensifies the sounds, whether this be dependent on inflammation of the organ itself or its membranes, or on general febrile disturbance previous to the occurrence of debility. But nervous excitement is the most powerful intensifier of the heart's sounds; it will even temporarily produce a loud first sound from a pure solidly hypertrophous left ventricle, and taken alone has much more frequently been known to render the sounds audible at a distance from the surface than any organic affection of the heart. I doubt if the latter, unaided by nervous excitement, be competent to produce the phenomenon.

The quantity of blood circulating through the heart also modifies the intensity of the first sound. If the quantity be considerable, the struggle of the ventricle to force it on is considerable and prolonged; but the sound does not gain, indeed rather loses, in loudness. When the quantity is small, on the contrary, there is no such struggle; the ventricles contract abruptly and sonorously, and probably, under these circumstances, collision of their opposite surfaces intensifies as well as modifies the quality of the first sound.

The sounds are *weakened* where the muscular structure of the heart is encroached upon by morbid infiltrations, or by its own disease. Softening, obesity, fatty degeneration, cancerous infiltration, atrophy with fibrous infiltration, all impair its force, both in its muscular and valvular elements, for obvious reasons. In dilatation with attenuation, the first sound, though clear, is feeble, —probably from conjoined change in the sarcous structure.

In simple, and still more in concentric, hypertrophy of the left ventricle, the first sound is weakened at the apex in, it may almost be said, the direct ratio of the increase in mass of the muscular substance and decrease of the cavity. In extreme cases, the first sound may be completely deficient over the ventricle, and perceptible only at the base, or towards the ensiform cartilage. The compact packing of the muscular structure interferes probably with the freedom of vibration necessary for the generation of sound, and the diminished size of the cavity obviously impedes the play of its parietes. Even the valvular action is diminished in scope, and while the mitral valve is of less proportional extent, it is often thicker than natural. The fibres, too, are deficient in tone,—they are less distinctly striated than natural, and non-striated fibres (it is said) appear among them: such fibres may be less sonorous in contraction. The entire mass of the ventricle, too, does not contract simultaneously.

Accumulations in the pericardium of fluid, of air, or of air and fluid combined, weaken the sounds by removing their source of production from the surface. I do not know clinically the effect of solid formations in the pericardium; they may be conceived to weaken the sounds in one way, and intensify them in another,—production and conduction.

Want of muscular and nervous tone generally weakens the first sound, as well as modifies its quality.

The *duration* of the first sound falls below the natural standard in dilatation without hypertrophy, or with attenuation, and under all circumstances which weaken the muscular, and throw into undue prominence the valvular, portion of the sound. The sound is lengthened more or less in hypertrophy, with moderate dilatation; and if, in addition, the mass of circulating blood be large, or if the aortic orifice be at all obstructed, the sound may be sufficiently prolonged to fill nearly two-thirds the period of each revolution of the heart.

The second sound is shortened by a thin papery state of the sygmoid valves and by thinness of the blood; its pitch rises under the same influences. The sound is lengthened, on the contrary, by thickening of the valves, and a loose inelastic condition of their texture. Probably, too, thickening of the non-striated muscular portion of the arterial walls (but I do not know this clinically) will lengthen their own systole, and hence, probably, the diastolic heart-sound. Another curious cause of prolongation of this sound will be by-and-bye described.

The points of maximum intensity of the heart's sounds are more liable to displacement by extrinsic than by intrinsic causes; the latter, however, in some forms influence them. Enlargements of the heart generally, whatever be their nature, *lower* the maximum point of the first sound. Simple, and *à fortiori* concentric, hypertrophy influence its position much less, in proportion to their mass, than the eccentric. Pericardial effusion, raising the apex, *raises* the point of maximum intensity of the first sound; and in some rare instances I have known this carried backwards, the sound being more distinct in the right vertebral groove than in the ordinary apex region. Hypertrophy of the right ventricle lowers its own first sound; of the left, carries its own immediately to the left, as well as depresses it. In all cases of depression of the first sound, the second is similarly affected, but to a less degree. Marked eccentric hypertrophy of the auricles would also probably lower the first more than the second sound; but I do not know this from experience.

The distance of the heart's sounds from the ear of the auscultator is very manifestly *increased* in cases of fluid and gaseous accumulation in the pericardium; but there is a source of fallacy here. The sounds are commonly listened to in the natural situation of the apex, and not in the neighbourhood of the nipple (fourth rib), whither it has been carried. Agglutination of the pericardial surfaces, on the contrary, brings the sounds *nearer* than natural to the ear, if not precisely at their maximum points, beyond these. Enlargement of the heart, sufficient to push away the left lung, will have a similar effect.

The first is more affected by these changes than the second sound.

The quality (and with this the pitch) of the heart's sounds is subject to serious modifications. The first becomes dull, muffled, toneless, and in some cases almost null, at the apex, where dense hypertrophy is conjoined with a thickened inelastic condition of the auricular valves. On the other hand, where the ventricular walls are thin and the valves natural, the first sound becomes more or less clear, flapping, or clicking, with raised pitch; if those walls be in a state of eccentric hypertrophy, and the valves somewhat thickened, the sound assumes a clanging character. At least, these statements are in accordance with the majority of results; but exceptions, explicable, perhaps, sometimes by the state of the heart's texture,—in others inexplicable,—pretty frequently occur. It would be difficult to describe or explain, for instance, the varieties of quality found in the first sound of soft, flabby, fatty hearts, with (practically speaking) sound valves. Clearness and elevation of pitch depend, as a rule, either on thinness of the muscular walls, or on predominance of the valvular element of the sound.

The first sound sometimes possesses a peculiar full-toned quality, without the least sharpness, while it is strongly accentuated at the commencement, and commonly prolonged: the nearest articulate symbol of the two sounds, under these circumstances, appears to be *b'oom-tup*, pronounced with strong emphasis on the *b*. I have observed this peculiarity in cases of eccentric hypertrophy of the left ventricle,—but without ascertaining the special condition on which it depends.

The quality of the first sound at the apex is sometimes sharply knocking; but with care this knocking quality is separated by the ear from the true heart-sound, and obviously depends on the impulse of the apex against the side,—but not necessarily against the inferior border of the fifth rib, as fancifully imagined by Hope. Knocking impulsive sound cannot be called an essential or even habitual attendant on any particular disease of the heart; nervous palpitation (especially if the edge of the lung be by some disease of its own carried unduly towards the left) will readily produce it in a sound organ. Thin-walled resonant chests supply it with greater ease than others; and morbid induration of the heart's apex, or calcification of the pericardium, will aid in intensifying it. The heaving and steadily pushing character of the impulse in simple hypertrophy prevents its occurrence in that disease: eccentric hypertrophy is the form of enlargement it most frequently accompanies.

The first sound is sometimes slightly rough, and approaching in quality to a murmur at the apex; it is, in fact, *murmurish*, without being actually converted into a murmur.* This may be (*a*) a persistent condition, observable week after week, while the patient remains under treatment; or (*b*) a temporary state, constantly noticeable for a few days, and then disappearing; or (*c*) a mere transient phenomenon, occurring with some, absent from other, beats of the heart. In the first case (*a*), it has appeared to me referrible to an incipient or slight amount of some one of the organic conditions which, carried further, produce a perfect systolic murmur; or, probably, sometimes to a buzzing, murmur-like quality in the muscular sound itself, produced by slow contraction of the fibres, or some special alteration of their texture. The second case (*b*) is exemplified by some *excessively rare* instances of acute rheumatism, where the systolic sound of the left apex, roughened and murmurish for a few days at the outset, then loses this quality, either permanently, or to resume it again at a later period in a more decided form.† Passing vascular roughness of the mitral, or even ventricular, endocardium, and imperfect closure of that valve from the influence of irritation, suggest themselves as possible causes of the phenomenon; but anatomical evidence is of course wanting on the point. In the third case (*c*), the peculiarity is caused either by coincidence of the respiratory sound with that of the heart,* or by rubbing of the apex against the pericardium,† or by movement of air in the adjoining lung-substance produced by the cardiac impulse, or (by far the most important cause, because the most likely to lead to error,) by a tendency to reduplication of the first sound.

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* In the case already referred to (p. 194), when the contraction of the abdominal muscles occurs in a slow vermicular manner, the sound is distinctly of murmur-like quality.

† Case of James Hayes. U. C. H., Oct. 1850.

The *second* sound at the base is rendered dull, and comparatively clanging, by fibro-fatty thickening, without insufficiency, of the sygmoid valves. Diminished elasticity of the arterial walls has a similar effect.

Like the first, the second sound may be murmurish, temporarily or permanently. Very trifling insufficiency will probably thus modify its quality; whether marked reticulation of the valves will suffice for the purpose, will hereafter be discussed. The most common cause of murmurishness in this sound is a tendency to reduplication.

The natural *accentuation* of the sounds, as shown in a previous page, is liable to numerous perversions; but as the accent falls on whichever sound is intensified, and mainly at the spot of intensification, repetition may be saved by referring the reader to the paragraphs on augmented intensity. When the heart is weak and flabby from organic change, or from want of tone (as in continued fever), there may be a total deficiency of accent on either sound at the apex ‡; the sounds resemble those of a vibrating pendulum.

Like the sounds, the *silences* vary in disease in relative duration. The first (or post-systolic) silence is lengthened by deficient elasticity of the arterial walls, whereby the recoil of the blood on the valves is sluggishly effected; so, too, whenever the first sound is disproportionately shortened (this is, perhaps, best observable in continued fever) the first silence is lengthened. The first silence is normally so short, that it is difficult to appreciate its decrease.

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* It is sometimes impossible to satisfy oneself on this point, unless by causing the patient to suspend his breathing.

† The more superficial character and the influence of change of posture will generally distinguish this variety from a true murmurishness.

‡ The second sound is in certain rare cases of mitral regurgitation, so intensified that the accent falls on it, even when ausculted at the apex.

The second (or post-diastolic) silence is lengthened in cases of advanced constriction of the mitral orifice; the process of filling the ventricle is laborious and slow, and hence the systole lags, as it were, behind its time. When the circulation is greatly slackened, the second silence is generally disproportionately prolonged.

The relationship of the sounds of the heart to the pulse varies in disease. In the normal state, the first sound is apparently synchronous with the diastole of the arch of the aorta, the pulmonary, carotid, and subclavian arteries; thenceforth, the further the vessel from the heart, the more distinct is the interval between the systolic sound of that organ and the arterial diastole. It is difficult to determine the possible length of interval consistent with health; but it may be affirmed, that if the diastole of the most distant vessels, as the posterior tibial and dorsal artery of the foot, is so much retarded as to become synchronous with the second sound, the state is morbid. This retardation, which was first detected by Dr. Henderson as an attendant on insufficiency of the aortic valves, may (with care) be detected in many, but unquestionably not in all, cases of that disease. Possibly where no morbid retardation can be discovered, the failure may depend, not on its absence, but on its being carried to such extremes, that the arterial pulse produced by one cardiac systole is nearly synchronous with the next. The only fact, however, I know of, supporting this idea, is, that it is in *extreme* cases of aortic regurgitation that the pulse seems occasionally to stand in normal relationship of time to the heart's systole. The same sign exists in attenuated dilatation of the left ventricle also. Again, in health, the frequency of the pulse and the length of the systolic sound vary inversely as each other: a frequent pulse is the index of a short first sound, and *vice versâ.* The same relationship holds good in some morbid states; for example, in anæmia and in the re-action after hæmorrhage: it is, on the other hand, occasionally perverted; the pulse may be infrequent, and the systolic sound short. In fatty degeneration, and in simple flabby softening of the heart, this perversion may sometimes (though rarely) be noticed.

The sounds of the heart are sometimes suspended for the precise length of time occupied by an ordinary revolution of the organ: they are said then to *intermit.* Very commonly such intermission recurs with considerable regularity; that is, after a fixed number of regular beats. Sometimes the systolic sound seems to *anticipate,* sometimes, on the contrary, to *hesitate* at, the proper moment of its occurrence—changes of rhythm closely connected with shortening or prolongation of the second silence. Sometimes a series of feeble and rapidly succeeding sounds follows others comparatively loud, slow, and deliberate; and there may be a certain uniformity in the number of each kind, and in the periods of their recurrence. Or the *irregularity* of the sounds (as of the contractions) may be complete, both in intensity and in rhythm, no two revolutions corresponding to each other in either character: there ceases to be any semblance of order in disorder. This excessive perversion exists in highly marked mitral contraction and regurgitation, in extreme softening (acute and chronic), fatty infiltration, acute destruction of a portion of a valve, or of chordæ tendineæ, rupture of these structures, formation of fibrinous coagula within the heart, and in a small proportion of cases of pericarditic effusion.

The natural correspondence in the number and time of cardiac systoles and arterial pulsations is habitually maintained, even when the rhythm of the heart's contractions is thus variously altered. If the left ventricle intermits, or anticipates, or hesitates, or becomes wholly irregular in its contractions, a precisely similar change occurs in the arterial pulses: the impulses of the connected tubes are the counterparts of those of the central organ. But, on the other hand, there may be a failure of this correspondence, not only when the heart's contractions are thus abnormal in rhythm, but even when they are in this respect normal. Thus two revolutions of the heart may correspond to a single radial pulse, the cardiac action and the pulse being perfectly regular in force and rhythm; or the pulse may be perfectly regular, and the heart's successive systoles somewhat unequal in force and duration, as in a case formerly recorded,* where eighty-eight systolic contractions produced forty-four radial pulsations. Here the rhythm of each pair of beats of the heart might be represented thus:—Systole = 5, diastole = 3; systole = 9, diastole = 4. It was the first of the two systoles that failed to affect the pulse at the wrist; and as there was no evidence of aortic or mitral disease, but merely of flabby enlargement, that systole may have been simply too weak to influence the distant vessels: the state was of temporary duration. I have observed a similar condition, persistent, but of less regular type, in cases of extreme contraction of the mitral orifice: under these circumstances, doubtless the systole occasionally takes place before the ventricle is supplied with blood to propel.

Again, in certain cases of utter irregularity of the sounds, there may be no traceable accordance between them and the force or rhythm of the pulse. This is, perhaps, best observable where the irregularity comes on suddenly from rupture of a valve, or accumulation of coagula in the cavities; but is occasionally met with in all the diseased states productive of irregularity.

The *number of sounds* attending each beat of the heart may vary, the arterial pulse holding its natural relationship to the systole. A single sound only may be heard, and this may be the first or the second; whichever sound be deficient in any particular spot, it may, or may not, be audible at some other part of the cardiac region. The first sound may be quasi-deficient at the left apex, when the conditions already described as weakening it, are carried to extremes; but it will then be found at the right apex, and at the base. So, again, the second sound may be quasi-deficient at the base from excessive feebleness, or from being covered by a prolonged systolic sound, or systolic murmur: but in the first case, excitement of the heart, increasing the energy of its contractions, will invigorate the sound, and in the second case, the sound will be heard at the right apex. Absolute deficiency of either sound (or of a murmur taking its place) has never fallen under my observation; in other words, neither systole nor diastole has ever been, in my experience, absolutely noiseless.

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* Clin. Lect., loc. cit. p. 443.

Reduplication of the first sound at the apex is not a very uncommon condition: the articulate symbol of the sounds may then be written *ŭbbŭp-dŭp* for the left apex, and *ŭppŭp-tŭp* for the right. Such slight accentuation as there is (and it is very slight) falls on the first of the three sounds. The first sound, while double at the left apex, may be natural and single at the right; or (what is more rare) the converse state may exist. In the great majority of cases, reduplication of the first sound audible at either apex, is audible at the base; on the other hand, reduplication at the base is commonly imperceptible at the apex.

Reduplication of the second sound at the base is the most common of this class of alterations of rhythm; the nearest symbol of the sounds then becomes *ŭp-tŭppŭp*, or *ŭp-tŭrrŭp*. Not only may the second sound, however, be pure and single at the apex, while thus double at the base, (indeed this is common,) but the reduplication may be audible or inaudible at both the pulmonary and aortic cartilages, or at either singly. Coexisting reduplications of both sounds are rare; they are more frequently audible at the mitral apex than elsewhere, when their symbol may be set down as *ŭbbŭp-tŭrrŭp*.

Sometimes a tendency exists to reduplication, without the sound becoming actually double; this state of things prolongs and gives a murmurish quality to the affected sound. The pitch of the two portions of the reduplicated sound is often dissimilar.

The essential cause of these various reduplications seems to be a want of synchronism between the actions of the two sides of the heart. If the facility with which the two ventricles fill with blood be unequal, they will probably differ somewhat in their time of contraction; certain conditions of the auriculo-ventricular valves on either side may bring their closure there, as it were, behind time; the production of a double systolic sound at the apex becomes thus readily intelligible: but the fact that such reduplication may be audible at one apex only, and perfectly inaudible at the base, is far from being easily explicable. It appears, too, from a case observed by M. Charcelay,[*] that the contraction of the auricles, when highly hypertrophous, may become sonorous, and so double the first sound; but it is scarcely necessary to add, that such mechanism is of singular rarity; nor does it appear probable that the characters of the reduplicate sound could, under the circumstances, resemble those ordinarily met with.[†] A simulated reduplication of the first sound may sometimes be produced by the knock of the heart against the side; but the least-practised ear will readily distinguish this from true *intra-cardiac* reduplication.

Non-synchronous tightening of the sygmoid valves, again, easily explains the reduplication of the second sound at the base; and may itself be referred to unequal elasticity in the coats of the aorta and pulmonary artery, stiffness of either set of valves, a material obstruction in the way of their closure, or any state of either ventricle rendering it slightly tardy in the propulsion of its blood into the artery beyond. But the same difficulty re-appears in accounting for the limitation of the phenomenon to certain points of the cardiac region.

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[*] Archives générales de Médicine, 1838, p. 393.

[†] I have recently met with a case in which an hypertrophous left auricle produced a distinct, even loud, *knocking* sound at the third left cartilage.—Geo. Smith, U. C. H., Nov. 1850.

In regard of diagnosis, it must be confessed, these reduplications are almost insignificant in the present state of knowledge. And for the following reasons:—reduplication is never (as far as I have observed) permanent and invariable; it occurs most commonly in hearts either healthy or functionally disordered only; less commonly in cases of slight organic affection; and with least frequency when serious valvular disease exists;* it is not connected, as a rule, with any particular form of disease; it comes and goes in the course of a few beats of the heart; sometimes disappears on change of posture, and is affected even by the act of respiration. The real interest of reduplications arises out of their bearing on the theory of the heart's sounds,—a fact of which the following illustrations are sufficient evidence.

The second sound may be continuously doubled at the base, and perfectly pure and single at the apex. How is this explicable on the sygmoid theory of the second sound? A double sound does not become single by conduction over so short a space.

The first sound may be single at the left apex and at the base, while it is distinctly reduplicate at the right apex. Here the ventricular and arterial portions of the first sound seem to be separated on the right side of the heart.†

The second sound may be double at the base, and single at the aortic, double at the pulmonary, cartilage (or *vice versâ*). This cannot arise from want of synchronism of the two sets of valves, but of the three divisions of one set.

The second sound may be single at the base and double at the left apex; now, according to the pure sygmoid theory, the arterial valves are the sole source of the second sound: how come the two sets to divide their compound sound at the apex? Splitting into two, as a result of conduction from the base to the apex by ventricles of different conducting powers, cannot be admitted; for the reduplication may be present at the left apex only, absent at the right. This is the strongest fact, I know of, in favour of the second sound being in some cases partly of ventricular origin.

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* It is fair to observe, however, that the existence of murmurs in such cases may be the real cause of reduplication not occurring, or not being heard.

† In one case (James Hayes, U. C. H., Oct. 17, 1850) where this form of reduplication was well marked, pericardial adhesions had recently formed.

II. *Adventitious Sounds or Murmurs.*—Sounds of adventitious origin and properties produced either within or on the surface of the heart, are termed Murmurs; according to their seat of production, they are divided into Endocardial and Pericardial.

A—*Endocardial Murmurs.*—The *special character* of all endocardial murmurs is, more or less, blowing. Their quality varies extremely, and they may be called simply blowing, grating, filing, rasping, sawing, whistling, cooing, &c., according to their greater or less similarity to those sounds. The simple blowing murmur, though itself presenting different degrees of harshness, is always soft in comparison with the filing, grating, rasping, and sawing varieties. In some cases they are tones, capable of musical notation. Endocardial murmurs never become audible to the individual himself, but they may in some instances be heard at short distances from the chest.* They are essentially *intermittent;* and, no matter what its duration be in relation to the heart's contractions, a single murmur is never sustained continuously, either in a uniform or remittent manner, through a series of beats. In point of absolute duration, they vary from a scarcely appreciable moment to two or three seconds: the latter amount of prolongation can only occur where the physical conditions are at once peculiarly favourable for sustainment of sound, and the action of the heart extremely slow. Their *pitch* varies by several notes, the lowest being, perhaps, represented by the whispered word *who*, the highest by *ss*; intermediate notes may be represented by whispering the word *awe* by inspiration, and the letter *r*, with various degrees of closeness of the isthmus of the throat: these are the suggestions of Dr. Hope. In point of apparent *distance from the surface*, they vary also: so deep, in some cases, as obviously to be weakened by distance; in others, they seem to originate directly under the integuments. The spots of *maximum force* of individual murmurs (the position of the heart being unchanged) are four,—a few lines above the left apex, just above the ensiform cartilage, at mid-sternum, on the level of the third interspace, and at the junction of the third left cartilage with the sternum. But these points of maximum force are liable to change, both from various mal-positions of the heart, and from alteration in the conducting qualities of the materials around it; the laws of transmission are then the same as in the case of the healthy sounds: a special cause, the direction of the current of blood, will be considered hereafter. An endocardial murmur once developed is habitually *persistent*, and attends every beat of the heart: however, weak systoles may fail to produce a murmur well marked with strong ones, tendency to syncope, general debility, and collapse, and the approach of death may prevent its production; and sometimes (especially when the cause of the murmur is pressure on an arterial orifice by a tumor) certain postures may annul a murmur completely. Murmurs habitually attain a higher type of harshness the longer they exist, though a fall in pitch may take place.

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* I have only once to my recollection met with this. In a case of cyanosis occurring in a child about six years old, a systolic rough blowing murmur could be heard at a distance of an inch or a little more from the surface.

The *rhythm* of murmurs may be considered in respect of the heart's contractions and of the heart's sounds. In respect of the former, they are said to be systolic and diastolic; in respect of the latter, synchronous with the first or second sounds. But they are not necessarily synchronous with either systole or diastole. They may be pre-systolic, systolic, and post-systolic; pre-diastolic, diastolic, and post-diastolic: they may occupy a portion only of either time, or fill this and the succeeding silence, and encroach upon the succeeding sound: this is common with systolic, rare with diastolic, murmurs. This arrangement is doubtless open to the charge of hyper-division; but as it positively has its foundation in correct clinical observation, it ought to be kept in view, although for ordinary purposes the simpler one is quite sufficient: there can be little doubt that many of the alleged failures of the rules for valvular diagnosis are traceable to inattention to these subdivisions of systolic and diastolic time. But murmurs are further divisible in respect of rhythm: whether systolic or diastolic, they may occur in the direction of the current of blood, or against it. Thus the systole of the left ventricle may produce a murmur at the aortic orifice with the current, or at the mitral orifice against it: hence a division of murmurs into direct and indirect,—which are also severally called, from their common causes, murmurs from constriction and from insufficiency or regurgitation.

The effect of murmurs on the sounds varies. A murmur may simply render a synchronous heart-sound obscure, at its commencement, at its close, or throughout its entire duration; or it may completely mask this by its intensity, and even similarly affect the succeeding sound; or it may prevent the natural heart-sound from being formed. Thus at the left apex a systolic murmur may completely drown a systolic sound, which is readily audible at the right apex and at the base. The systolic sound is not masked there, but really deficient, when both auricular orifices are not in a condition to produce natural sound, but murmur alone, and the arterial (or basic) portion of the first sound is feeble, or itself converted into a murmur.

When, as is very common, both murmur and sound are distinguishable coetaneously, the state may be called one of pseudo-reduplication. But true reduplication of a murmur is so excessively rare, that I remember to have met with but two examples of the fact: one, basic and diastolic, must have been in the aorta alone, for the pulmonary artery was unaffected; of the other, systolic at the left apex, I had no opportunity of examining the mechanism;—it might have been mitral and tricuspid together, or it might have depended on co-existing deep-seated venous hum,—in which case, of course, it was a false reduplication only. I once heard a post-diastolic basic murmur and sound in a rheumatic woman, in whom the doubling appeared to come from the set of sygmoid valves, which gave the murmur, acting after the set which gave the sound.

When endocardial murmurs have existed during life, structural changes to explain them are found, or are not found, after death: hence their division into *organic* and *inorganic*.

(a) *Organic* murmurs are essentially connected with such alterations of the orifices or of the valves, as, while they lead to constriction or imperfect closure of the orifices, cause unnatural friction of the blood and surface. The chief of these alterations are: simple constriction, or constriction with thickening, hardness, rigidity, calcification, warty or other excrescences from, or even simple inflammatory loss of polish and roughness of, the valves;—simple insufficiency of the valves to close a widened orifice, themselves not having grown *pari passu* with the widening, or such insufficiency depending on the various diseases of the valves just enumerated, or depending on shortening and thickening of the chordæ tendineæ, or on atrophy or contraction of the columnæ carneæ, or on puckering of valves, or adhesion of the divisions of a valve *inter se*, or to the adjacent surface;—excrescences or other thickening or calcification of the valves, without either insufficiency or constriction;—or unnatural communication between the different compartments of the heart, or between these and the arteries or some adventitious cavity. Besides, without distinct alteration of the orifices or valves, mere vascular roughness of the ventricular endocardium probably suffices to affect the purity of the sounds, when the current bears, especially towards the arterial orifices, on such a roughened spot. Coagula among the columnæ, or a polypoid body hanging from the neighbourhood of the valves, will have a similar effect.

Physically speaking, then, there appear pure constrictions of natural orifices, pure widenings of orifices, and pure roughness of surfaces, to explain the *mechanism* of cardiac murmurs, in some cases,—the last associated with either of the two former, in other cases. Now pure constriction and pure roughness are positively capable of producing murmur: this is matter of experimental demonstration. It is not so easy to conceive how pure widening of an orifice can produce murmur: in the case of the tricuspid and mitral orifices, the regurgitation from such a physical cause may intelligibly produce murmur by the collision of direct and indirect blood-currents coming from and going back into the auricle: it is not so easily intelligible how a direct current can produce murmur through a simply widened orifice; but the rippling of the stream, produced by the change of calibre, seems, from the experiments of Dr. Corrigan, sufficient, if the current be strong, to produce it.

Mere alteration in the direction of the current, of a kind to throw the blood obliquely against an orifice, instead of carrying it directly through, will theoretically generate murmur. Probably this plays a part in many direct valvular murmurs; but as dilatation of the ventricles renders them more spherical and less convergent to their arterial outlets, it has been urged by Dr. Blackiston and others that such state must produce murmur if hypertrophy co-exist,—that it actually is heard frequently under the circumstances; and, when wanting, its deficiency proves that the muscular energy is impaired by disease. Hypertrophy with dilatation of a cavity, if its arterial outlet remain undilated, puts that outlet relatively in a state of coarctation: hence, too, may arise a murmur. I will return to these points with the subject of eccentric hypertrophy.

It was suggested by M. Martin-Solon that the pressure of the heart and great vessels by abundant pericardial effusion might cause blowing murmur;—in a case of the kind, murmur was well marked in recumbency, disappearing when the patient stood up.*

The *properties of murmurs vary greatly with the conditions of the fundamental cause producing them,* and even with some conditions independent of this. Thus the force, loudness, or

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* Journal Hebdomadaire, ix. 457.

intensity of a murmur increases with the vicinity of its origin to the surface,—the density and hardness of the heart itself, and of the textures lying between the heart and surface,—the force and velocity of the current, and, through this, the amount of narrowing at an orifice, and the volume of blood propelled through the obstruction. Excite a tranquil heart, and a murmur, previously almost inaudible, becomes distinct; weaken the energy of cardiac contraction by digitalis or aconite, and the converse result follows.*

The *quality* varies with the character of the surface over which the blood passes;—harsh and rough, if the surface be sharply uneven; soft, if smooth and merely constricted. But this influence is greatly modified, and may be actually reversed, by changes in the celerity of the circulation. The quality will also be materially affected by the condition of the intervening structures: if these be soft, the sound will be softened in quality; if hard, hardened. A musical quality is sometimes given when prominent spiculæ, of vibratile character, project into the current; and also when rigid vibratile edges bound a narrow chink-like opening.

The *duration* of murmurs will increase directly as the extent of surface in the condition to afford them, the amount of difficulty to struggle against, the quantity of blood, and the slowness of the circulation. If the structures intervening between the seat of murmur and the surface be imperfect conductors, the audible sound will be shortened,—its termination will be lost through imperfect conduction.

The *pitch* of a murmur is more under the influence of the size of the orifice through which the sonorous stream passes, than anything else: the smaller the orifice, the higher the pitch. But it is also raised by the tension of the walls of that orifice, and the thinness of the blood. The velocity of the current does not influence it, Dr. Blackiston urges, unless, as in blowing with a flute, the harmonic be elicited; but unless the edges of the diseased orifice be of such rigidity as to simulate the embouchure of the flute, it appears to me the two cases are not comparable. The distance of the site of production of murmurs from the surface has no influence on their pitch. Hope erroneously taught the contrary.*

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* In a woman now under observation (Emma Powell, U. C. H., Dec. 1850,) the systoles are so unequal in force that while some give a strong radial pulse, others produce none; the strong systoles are attended with systolic basic murmur, the weak ones with none.

Hence it appears that the properties of a murmur, as caught by the ear, are every one of them, singly, of complex mechanism,—and herein appears an easy clue to the absolute failure of all clinical attempts to establish the *precise amount and character* of the organic changes in a set of valves from the consideration of any one property (such as roughness) of the murmur they generate. Every necessary organic condition of a harsh murmur may be present, and yet the resulting murmur be soft, if the current be feeble,—nay murmur may be wholly deficient.†

(b.) *Inorganic Endocardial Murmurs.*—Endocardial murmurs, that cannot be traced to any organic cause after death, or that disappear so completely and permanently during life as to preclude the idea of structural change in their production, are termed inorganic. They are divisible into two sub-classes—murmurs of blood origin, and of dynamic origin.

1. *Cardiac Blood-murmurs.*—An intra-cardiac blood-murmur is of moderate or very slight intensity, commonly of medium or low pitch, short or moderately prolonged, of whiffing quality, very

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* To whatever distance a note continues audible, it remains the same note as at the spot of its generation; were it otherwise, each individual of the audience at the opera, for example, would hear a different score from his neighbours.

† I have, on the other hand, occasionally known a murmur, when audible in a calm state of the circulation, disappear during palpitation. I have only observed this in some cases of mitral regurgitation. Can it depend on irregular contraction of the wall of the ventricle allowing of such slight and feeble regurgitation that morbid sound cannot be generated?

easily rendered temporarily harsh by excitement of the heart, and modified in intensity by certain changes of posture. This murmur is, as far as I have observed, invariably basic in seat and systolic in time, produced at the orifices of the aorta and of the pulmonary artery,—with a force at each proportional to the power of its communicating ventricle; scarcely conducted along the aorta at all; frequently audible, on the contrary, at the second left (or pulmonary) cartilage; only in exceptional cases audible below the nipple; and never, within my experience, perceptible as far as the left apex. The site and rhythm of these murmurs, excluding all those of diastolic time and seat at either apex, are of great value in their distinction. To their quality and pitch I attach but moderate importance; for organic murmurs may be soft, and inorganic ones are not very unfrequently rather harsh; while the latter may be shrill, whistling, and of high pitch, and the former are of course frequently of low pitch. Unfortunately there is no character in a systolic basic blood-murmur which positively proves its nature, and distinguishes it, *under all circumstances*, from one of organic source. The distinction is often rather to be made through the absence or presence of venous hum (I do not remember ever to have observed an intra-cardiac spanaemic murmur unattended with venous hum) and the course and duration of the phenomenon, than through its own immediate characters: *permanent* harshness and high pitch are never associated with murmur of blood-origin. If the arteries be the seat, extensively, of strong blowing murmur, without artificial pressure (as, for example, the arch of the aorta, the innominate and subclavian arteries,) a co-existing systolic murmur at the mid-sternal base is, in part at least, inorganic; but, on the other hand, the cardiac murmur may be truly inorganic, and yet the arteries be perfectly free from abnormal sonorousness.

The morbid state of the blood which acts as the most frequent cause of these murmurs is spanaemia,—whether it be that of chlorosis, of malaria, of starvation, of deficient insolation, of hæmorrhage and over-venæsection, of carcinoma, of convalescence from acute diseases, &c. I have occasionally observed it in uræmia; so, too, it occasionally occurs (though to a very slight amount) in the hypinosis of continued fever and the exanthemata, and in the hyperinosis of pneumonia and acute rheumatism, under circumstances excluding more or less positively its dependence on endocarditis. It has been affirmed that plethora, rendering the quantity of blood too great for the cavities of the heart, produces murmur within it: confirmation of the statement is wanting; more especially if we agree with those chemists who maintain that the relative quantity of red corpuscles is increased in plethora.

The mechanism of these blood-murmurs will be considered with that of the venous class.

2. *Dynamic Cardiac Murmurs.*—Under this head murmurs occurring in the heart, through some abnormal state of its action, range themselves. Violent excitement of the organ, whether it act merely by increasing the force of the current, or by disturbing its direction, occasionally produces for the time systolic murmur at the base. I have observed this not only in hysterical females (who, though of florid countenance, might have been slightly spanaemic), but in males with palpitating heart. If the organ be the subject of dilated hypertrophy, palpitation is sometimes, but certainly not always, attended with the same murmur. In hypertrophy with dilatation there is possibly another dynamic source of systolic murmur: from the altered form of the cavities of the ventricles, their contained blood is probably propelled against the edges of the arterial orifices, instead of directly through them; and this misdirection of the current may, very possibly, generate murmur.

The heart may, probably, also undergo dynamic changes interfering with the closure of its valves, and giving rise to murmurs of the regurgitant class. A systolic murmur at the left apex is occasionally audible in chorea, disappearing as the disease disappears,—which cannot be referred to inflammation or organic disease of the mitral valve; which cannot be of blood-origin, but which does seem plausibly ascribable to disordered action of the muscular apparatus connected with the valve.* The same kind of disorder may conceivably be the cause of certain mitral regurgitant murmurs, attending dilated hypertrophy of the left ventricle, which disappear under treatment (*e. g.* case of Bonsey, U. C. H. Oct. 1850).

In seeking the causes and seat of any given endocardial murmur, the essential points in the inquiry are its relationship to the systole or diastole, and the spot of its maximum intensity on the surface of the chest. Subsidiary conditions of great importance are the direction of transmission, duration, clinical progress, quality, and pitch of the murmur; and, beyond itself, the state of the heart's natural sounds, and the presence or absence of certain audible phenomena in the arteries and veins, or both.

Each orifice of the heart may be the seat of two murmurs, constrictive and regurgitant,—with, or against, the current: the total number of cardiac murmurs connected with the orifices, therefore, reaches eight. The essential characters of these may be briefly set down as follows :—

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* If the heart be removed from the body, an auricle cut away, the artery of the same side tied, and the cavity of the ventricle filled with fluid; and if, then, a stream of water be directed upon the auriculo-ventricular valve, this rapidly closes. From this experiment of Baumgarten, the inference has been drawn that the auriculo-ventricular valves are closed by the systole of the auricles, prior to the systole of the ventricles, and that the closure is not in anywise influenced by the musculi papillares, but is much facilitated by the specific lightness of the valves themselves. The multitude of points in which the experiment fails to imitate the natural state of things in the living and contracting heart, utterly invalidates, in my opinion, the conclusion it has been forced to furnish. But, even granting that this inference be sound in *physiology*, the additional assumption of M. Hamernik, that *morbid* conditions of the muscular structure of the heart can have no effect in promoting closure of the valves, is a palpable *non sequitur*. What! suppose that (*inter alia similia*) the papillary muscles are shortened, puckered, dwindled in muscular texture, and infiltrated with pseudo-fibrous induration-matter (as they occasionally are), will not this state somewhat, at least, interfere with the motion of the valves? We shall next be told that shortening and thickening of the chordæ tendineæ produces not the least embarrassment in the play of the valves.

a. A *systolic* murmur of maximum force at, and immediately above, or to the outside of, *the left apex,*—but faintly audible, or wholly inaudible, at the right apex (say the ensiform cartilage), the mid-sternal base,* and the pulmonary and aortic cartilages, —more or less clearly audible about and within the inferior angle of the left scapula, and beside the dorsal vertebræ from the sixth to the ninth, is essentially characteristic of *regurgitation through the mitral orifice* at the moment of the ventricular systole. This regurgitation may be the result of inefficiency of the valve, produced by chronic changes of structure (its common cause),—or by enlargement of the orifice without coeval growth of the valve (a *very* rare cause),—or occur from non-closure in acute endocarditis, in consequence of roughness of the edges of the valves and shortening of the chordæ tendineæ,—or, perhaps, from disordered action of the columnæ carneæ. But it is not absolutely pathognomonic of such regurgitation; for, in some very rare cases, fibrinous coagula amid the columnæ carneæ, near the valvular portion of the ventricle, and vegetations on the ventricular surface of the valve, have produced a murmur of this rhythm and site.

Of very variable quality, this murmur is rarely of high pitch, generally oscillating between whispered *who* and *rr.* I have known it sharply whistling (as of the wind through a key-hole), and musically pitched in a high key. Once established, it is, as a rule, permanent; but when traceable to irregular muscular action (as in chorea) it may wholly disappear. If the cause of non-occlusion act intermittently, the murmur will be present at some moments, absent at others. This I once observed in an adult male, in whom a body about the size of a large pea was suspended by a thread-like peduncle from the larger division of the valve, in such manner that it might or might not, according to accidental circumstances, have fallen within the orifice, and impeded its closure.

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* If the stethoscope be carried gradually upwards and inwards from the maximum point towards the third cartilage, it will be found that at a certain point, defined with singular sharpness, the characters of the murmur completely change: in intensity, it falls to a third; in roughness, loses greatly; in openness, gains.

A mitral regurgitant murmur may completely or partially cover the first sound at the left apex. This first sound may have its natural characters in perfection at the base and at the tricuspid apex; but when intense the murmur may partially obscure the sound in both these places by conduction.

In the healthy state, the second sound is more strongly accentuated in the aorta than in the pulmonary artery. The reverse, according to Skoda, holds in cases of mitral regurgitation; and the peculiarity is explicable thus:—With every systole some blood is forced into the left auricle; that auricle, the pulmonary veins, and pulmonary artery quickly become overstretched, and the right heart requires greater effort to force the blood into the over-filled vessels; the pulmonary artery consequently presses with increased force on its column of blood, and hence intensifies its own portion of the second sound. And, as a corollary, he holds that the absence or presence of this intensification will distinguish systolic murmur at the left apex, caused by regurgitation, from that caused by friction of the blood against roughnesses in the ventricle. I do not believe (although it certainly does exist in some cases) that any such implicit confidence can be placed in this sign. I have known it positively wanting, even when there was no obvious tricuspid regurgitation to afford a plausible explanation (through diminished current) of its deficiency; and it may exist independently of mitral regurgitation,—for instance, in hypertrophy of the right ventricle. It seems to me sometimes, too, only a pseudo-intensification, from real weakening of the aortic second sound through the lessened current and diminished caliber of that vessel that follow on long-continued mitral regurgitation. Besides, I have not found thickening or enlargement of the pulmonary artery in such connection with mitral regurgitation, as ought to obtain, were the theory described wholly accurate,—the more so, as thickening and enlargement of the left auricle really do exist in a fair proportion of these cases.

A murmur with the characters now described is the most common of the organic class,—is rarely of functional, and never, so far as I have seen, of purely blood-origin; and is always connected with the mitral valve, orifice, or neighbouring portion of the left ventricle.

β. A *systolic* murmur of maximum force *immediately above, or at the ensiform cartilage*, inaudible, or nearly so, at the left apex, and very faintly, if at all, perceptible in the left vertebral groove opposite the lower angle of the scapula, originates in the *right ventricle.* In the great majority of cases arising from *tricuspid regurgitation*, it may by possibility depend on sharp collision of blood among thickened and roughened chordæ tendineæ. In the former case, distension and pulsation of the auricle, vena cava, innominate and jugular veins is habitually present (that of the latter *visible*); but, as will hereafter be shown, the conclusion of M. Skoda, that, wherever such pulsation is absent, the murmur under consideration may be pronounced not to depend on regurgitation, is at variance with facts.

This tricuspid murmur is generally soft, and of low pitch, rarely masks the systolic sound completely, is of rarer occurrence than tricuspid regurgitation itself, and is probably not always detected when it exists. It is, absolutely speaking, rare, because regurgitation often occurs from insufficiency, without morbid change, of the valves, and because the back current is often not forcible enough for the production of a murmur. On the other hand, it escapes detection, because it is often covered by a powerful mitral murmur, and in some cases impaired in distinctness by deep-seated venous hum. In rare instances, where a mitral and tricuspid murmur coexist, a spot may be found midway between their maxima points, where there is comparatively little murmur.

γ. A *systolic* murmur, of maximum force, *at mid-sternum, opposite the third interspace* (or, it may be, the upper part of the fourth rib), abruptly losing force between this point and the left apex, where it may be almost inaudible, faintly perceptible at the second left cartilage, clearly audible at the second right cartilage, the notch of the sternum, and the left vertebral groove, opposite the second, third, and fourth vertebræ, thence rapidly losing strength downwards, and disappearing about the sixth, originates at the *aortic orifice*. It habitually signifies simple or rough constriction of that orifice, and in rare cases has been traced to fibrinous coagula impeding the egress of the blood. But cardiac murmur, very closely simulating this type, may be produced in cyanosis and aneurism of the heart and aorta. This murmur is commonly of high pitch, loud, prolonged, and harsh; an hypertrophous ventricle concurring, increases its intensity, and may give it a drawling prolonged character, if the sides of the orifice be much contracted.

This is, besides, as already explained, the commonest of intra-cardiac blood-murmurs.

Although audible at the aortic cartilage, it is distinctly fainter there than at the base; if as marked, and *à fortiori* if more marked there than opposite the valves themselves, the arch of the aorta itself adds to the murmur. The same inference is justified by any notable difference in pitch in the two spots.

δ. A *systolic murmur*, of maximum force *at the sternal edge of the third left cartilage*, or a little lower down, audible at the pulmonary cartilage, almost inaudible at the aortic cartilage and at the apex, and imperceptible in the back, indicates *obstruction at the orifice of the pulmonary artery*, simple roughness in its valves, or (as noticed by Dr. Elliotson), pressure on the vessel by adventitious masses in the pericardium. All these causes combined, however, are so unusual, that few persons have actually met with such a murmur (some simulating murmurs will be described hereafter). I have only observed one of the kind; and as, in this instance, there was no *post-mortem* examination, its site cannot be held to have been certain, seeing that we have no actual experience of its characters.

ε. *A diastolic murmur*, of maximum force, *immediately above and about the left apex*, and conducted on the same principle (though less extensively) as the systolic murmur of the same seat, indicates *obstructive narrowing of the mitral orifice*, or simple roughness of the auricular surface of the mitral valve, or both. Skoda affirms that murmurs of the two sources may be distinguished by the state of the second sound in the pulmonary artery: it will be intensified in the case of narrowing; unaffected in that of simple roughness, unless accidental causes of intensification be present. The least reflection on the infrequency of direct mitral murmur, and on its frequent accompaniment, when present, by *regurgitant* mitral disease, will show how hazardous the assertion of Skoda is;—it has, indeed, all the aspect of an *à priori* one.

I have never heard this murmur of great intensity, nor high pitch; it is, however, sometimes prolonged. It is rarely loud enough to cover the second sound completely, even at the left apex.

I have spoken of this murmur as diastolic in rhythm; but in point of fact it is rather post-diastolic or præ-systolic, than precisely coincident with the diastole.

This murmur is not very unfrequently wanting, where constriction is found after death. Sometimes this may be fairly referred to the weakness of the auricular systole and smoothness of the constricted orifice; where the constriction is slight, there will be but slight friction too. When deficient, as it has been, in cases of extensive contraction, Hope thought the deficiency depended on the very fact of the extreme smallness,—an explanation not over plausible.

I have known this murmur come and go from day to day in a case where the mitral orifice was very greatly contracted and rigid.*

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* U. C. H., Kernis (Females), vol. ii. p. 240.

• ζ. *A diastolic murmur,* of maximum force, at *the ensiform cartilage,* the most faintly audible at the left apex, and inaudible at the base, would probably indicate *tricuspid narrowing,* were there a hypertrophous auricle behind that orifice, to give force to the current; but I know nothing of the murmur by experience.

η. A *diastolic murmur,* of maximum force *at mid-sternum, opposite the third interspace or fourth cartilage,* conducted (with some exceptions, to be mentioned presently,) on the same principle as the systolic murmur of the same site, indicates *regurgitation through the aortic orifice.*

This murmur may be heard with almost as much intensity about the ensiform cartilage as opposite the third interspace—in this point of view differing materially from constrictive aortic murmur: the more distant conveyance downwards in the former case probably depends on the downward direction of the current producing the murmur. So marked is the fact, that unless with care the murmur of aortic regurgitation might be mistaken for one of tricuspid constriction. It is remarkable that the conduction is more perfect towards the right than the left apex,—consequently not (as might have been supposed) in the direction of the communicating ventricle: concerning this point I find my experience at variance with the statement of Hope.

The second sound of the heart may be covered completely at the maximum point of the murmur; or it may be heard at the beginning of, during, or at the close of this. In the first case, the valves are utterly incompetent: in the varieties of the second case, one division of the valve may flap naturally, or all three imperfectly, and so produce an imperfect second sound; or the second sound heard may be wholly that of the pulmonary valves.

Unless the murmur be of very great intensity, the second sound may be well heard at the left apex. The causes of this have already (p. 197) been inquired into.

Aortic regurgitant murmur is usually of aspired blowing quality, sometimes almost hissing, rarely rough, weak as a rule (though I have known it extremely loud, and it is said to have been heard at a little distance from the surface), and of considerable duration, habitually filling the post-diastolic silence.

The causes of this regurgitation are the ordinary ones producing incapacity of valves; I have known sudden rupture of one of the sygmoid valves produce it (Case of Gordelier, Cons. Hosp. Males, vol. i.). Reticulation of the valves carried to extremes, a conceivable source of murmur, I have never actually known to generate it; but perforative destruction on a large scale will of course do so. Mere incapacity of valves, healthy in themselves, but too small to fill the widened mouth of the aorta, has in rare instances produced this murmur. In rare cases it is developed during the *acute* period of endocarditis.

θ. From experiments on animals, it appears that a *diastolic* soft prolonged murmur, audible down the ventricle, may be produced by artificially rendering the *pulmonary valves insufficient.* But this regurgitant murmur is of excessive rarity in man: I have known an aortic regurgitant murmur more distinct at the second left than the second right cartilage—an obvious source of fallacy.

In respect of relative frequency, I should be disposed to place intra-cardiac murmurs of *organic* origin in the following order, commencing with the most common:—mitral regurgitant; aortic constrictive; aortic regurgitant; mitral constrictive; tricuspid regurgitant; pulmonary constrictive; pulmonary regurgitant; tricuspid constrictive.

These murmurs may be variously associated; the following combinations are those I have observed most frequently:—aortic constrictive and mitral regurgitant; aortic constrictive and regurgitant; mitral regurgitant and aortic regurgitant; mitral regurgitant, aortic constrictive and regurgitant; mitral regurgitant and obstructive; mitral regurgitant and tricuspid regurgitant; mitral regurgitant and constrictive, aortic constrictive and tricuspid regurgitant.

No one organic murmur involves, as matter of necessity, the presence of another; a direct murmur may exist at any valve, and an indirect be absent, and *vice versâ*.

When two murmurs co-exist at the same orifice, they are readily distinguishable by their rhythm, their quality, their pitch, and by their character of aspiration, or the contrary. I have great difficulty in believing with Skoda that mitral systolic and diastolic murmurs can so run into each other, that they shall appear to be one murmur of like quality from first to last.

B.—*Pericardial Murmurs.*—Pericardial murmurs are divisible, in respect of quality and of the mechanism of their production, into four chief species: (*a*) Friction or attrition-murmurs; (*b*) continuous murmurs; (*c*) clicking murmurs; (*d*) murmurs produced by bending of layers of exudation-matter. Plastic matter, with or without liquid, is the essential statical condition of them all,—movement their essential dynamic element.

(*a*) Attrition-murmurs, all of them, more or less, distinctly suggestive of rubbing of surfaces of variable character against each other, occur in a greater number of varieties even than pleural friction sounds. They resemble, for instance, *grazing, coarsely rubbing, grating, scratching, creaking, squeaking, prolonged whistling,* &c., sounds. Traceable as all these varieties commonly are to collision of surfaces roughened with lymph, the interest attached to their distinction must turn mainly on their being severally connected with some particular state of that lymph. Now, experience does not show any such necessary connection; though it be true, for the most part, that at the very earliest period the murmur is of grazing quality (resembling the sound produced by rubbing pieces of silk together), and that as the exudation hardens, and gathers into irregular peaked elevations, the quality becomes coarsely rubbing, grating, creaking, this sequence of changes is subject to constant exceptions. Creaking friction-sound, so loud as to be audible *three inches from the end of the stethoscope applied to the surface in the ordinary way,* may depend on tough exudation-matter with fine rough elevations; and I have noticed distinct, though slight, creaking quality, when the exudation-matter was found to be of almost creamy softness. Dr. Taylor,* too, has known signs of moderate liquid effusion coexist with friction-sound of this quality,—a fact showing that peculiarly rough attrition is not required for its production.

(*b*) When exudation-matter and fluid coexist, the former imprisoning the latter in its meshes, the heart's action produces a peculiar continuous rumbling (just such as, we find, occasionally occurs in the pleura) or squashy churning sound. This is rare; but, once heard, can never be forgotten.

(*c*) Occasionally sounds are heard of peculiar clicking character (only one or two, with each beat of the heart), which are only distinguishable, at the time, from modifications of the valvular sounds, by their non-synchronism with these, and by the extreme irregularity of their occurrence. I have satisfactorily traced these clicks to the pericardium, and further, in all probability, to the separation (without attrition) of surfaces glued together with exudation-matter.

(*d*) It has appeared to me that sound is sometimes generated in layers of firm false membrane, though so perfectly agglutinated together that attrition or separation of the apposed surfaces is physically impossible. The quality under such circumstances is probably variable: in the only positive instance of the kind which I have observed, it was faintly creaking. The bending and crumpling of tough false membrane may conceivably generate such sound.

The varieties of pericardial murmur may be heard over all parts of the cardiac region from the roots of the large vessels to the apex. But I have never detected the churning variety except about the apex, nor the clicking variety except in the site of the large vessels. The finest shades of grazing sound are most common behind the sternum. Different qualities of murmur may be heard at one and the same time in some cases over different parts of the heart; but this is not common. As a rule, friction-sound is most clearly and frequently detected below the third interspace, probably simply because, below that part, there is little lung intervening between the pericardium and surface. In rare instances, friction-sound in the pericardium is audible in the back, between the scapulæ and the spine (I mean in cases where mere intensity of sound cannot be held to explain the fact). It has been found single in the back, too, while double in front.* It seems sufficiently likely that in some, at least, of the cases where it is alleged to have been confined altogether to the back, the friction heard may have been produced in an inflamed pleura by the cardiac impulse. Pericardial friction is usually abruptly limited.

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* Brit. and For. Med. Rev., vol. xxiv.

Pericardial murmurs vary widely in intensity; so delicate in some instances, especially when of grazing or clicking quality, that the closest attention is required for their detection; in other cases they may be heard in the posterior and lateral regions of the chest, and even, as already mentioned, at a considerable distance from the surface in the præcordial region. On the whole, their intensity is greater than that of pleural-friction sounds; a fact accounted for by the comparative abruptness and energy of the motion inducing them.

When the entire cardiac surface is the seat of friction-murmurs, the maximum amount of sound exists, according to some writers, about the nipple; to others, behind the sternum. I am satisfied no rule of the kind can be established: I have known the point of maximum intensity change from the fourth interspace to the fifth, thence to the ensiform cartilage, and thence to the nipple, within twenty-four hours. Neither can any particular part of the cardiac surface be fixed on as the absolute seat of loudest friction-sound; it is sometimes, though rarely, excessively loud even about the base.

Pericardial friction may accompany both the systole and diastole, or either singly; its co-existence with the systole alone is not rare; with the diastole alone, infrequent. When of regular rhythm, the friction-murmur falls a little after the corresponding valvular sound. But such regularity as this is the exception, not the rule; the friction-sound may be very distinct during the post-diastolic silence. Friction coexistent with the systole is generally, but by no means constantly, sharper and louder than with the diastole.

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* Brit. and For. Med. Rev., loc. cit.

Pericardial murmurs are so deficient in tone, that their pitch can scarcely be estimated even rudely. Still they do embrace in their different varieties a certain compass of sounds. However, no practical hint, that I know of, is derived from their pitch, except that, generally speaking, the higher this is, the drier and rougher the material of attrition. The pitch of a pericardial murmur may sometimes be raised by pressure with the solid stethoscope.

Pericardial murmurs are, as a rule, distinguished by their superficial character: they appear to be produced immediately underneath the integuments. But if the physical cause of the murmur be placed beyond the limits of the triangular portion of the heart, uncovered by lung, they lose this character, and seem of more or less deep origin. This is sometimes particularly observable about the large vessels (before any effusion has occurred to push the edges of the lungs aside), when their apparent depth contributes to assimilate them in certain varieties to valvular murmurs.*

Various circumstances modify the intensity and superficial extent of these murmurs; the physical condition of the pericardium itself remains the same. The most important of these is the energy of the heart's action: the greater this, the louder the friction sound. The weakness immediately ensuing on blood-letting, approaching syncope, the action of digitalis and aconite, all lower its intensity: if successive systoles be of very unequal force, friction, absent with one, is present with another, impulse. An hypertrophous and dilated heart gives, *cæteris paribus*, the maximum quantum of attrition murmur. Attrition murmurs are more marked in expiration than in inspiration; and when the trunk is bent forwards, than in the recumbent position. Moderate pressure, especially with the solid stethoscope, commonly intensifies them; strong pressure may mask them completely.

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* Skoda attempts to show that the distinction of sounds, produced deeply and superficially, is a delusion. If he place a stethoscope on the chest of a dead body, and listen to the sounds produced by tapping different parts of the parietes, near and distant, he will, henceforth, probably relinquish the attempt.

The duration of friction-murmur varies very greatly. I have known it appear and disappear, finally, within six hours,—of the grazing variety, it is true, but still of unmistakeable characters. In illustration of the other extreme, I may refer to a case in which it continued audible for upwards of three months, especially at the lower part of the sternum.*

Pericardial friction may appear very rapidly after the cause of inflammation has acted. Thus, in the remarkable case at University College Hospital where fatal perforation of the œsophagus and pericardium was produced in the attempt to swallow a sword, friction was detected by my then Clinical Assistant, Mr. Tidmas, thirty minutes after the accident.

The total disappearance of pericardial murmur may, if it be slight in amount, almost immediately follow general or local bleeding; this disappearance may be only transitory, however, from weak action of the heart: or a pericardial murmur may abruptly disappear from rapid agglutination of the entire of the affected surfaces. I have known it impossible to find a vestige of friction in a case where, six hours before, the *entire* cardiac region was the seat of intense rubbing sound. But, generally speaking, the progress of agglutination is materially slower than this, and for several days one or more limited spots may be found, where the collision continues sonorous (sometimes in the churning variety), after the adhering process has commenced. The sudden cessation of friction-sound, through absorption of exudation-matter, is less easily conceived; and where such cessation occurs, independently of agglutination (or liquid effusion), the inflammation had probably produced very little indeed of that matter.

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* Case of Kennedy, U. C. H. Case Books, vol. i. p. 67. First heard on the 27th Oct.; it was last heard on the 5th Feb. following, long after the man's discharge and apparent restoration to health. How much longer it may have continued I know not, as the man was not seen again.

In the majority of cases where friction abruptly disappears, the change depends on fluid effusion, separating the pericardial surfaces from each other: it is consequently oftener an evil, than a good, sign. With accidental circumstances, such as the size of the heart, conformation of the chest, absence or presence of adjoining pleural adhesions, &c., the amount of liquid required to annul friction-sound varies. A case of Dr. Taylor's (loc. cit.) shows that friction may continue when eight ounces of fluid (or thereabouts) have accumulated: but generally less removes it. Disappearing with the occurrence of effusion, friction-sound pretty frequently returns when the fluid is absorbed: this returning (redux) friction generally, but not always, appears first about the great vessels and base. Pleuritic friction, we have seen, is more commonly caught at the redux period; pericardial, certainly, at the outset of the inflammation. The second disappearance of friction may be abrupt, slow, and gradual, irregular with recurrences, or rapid over the heart generally, while in a single spot or two (most commonly either at the apex or about the great vessels) some form of the murmur remains for a time.

Friction in the pericardium signifies inflammation of the membrane. It is exceedingly probable that mere dryness of the surfaces will suffice to produce the grazing variety. I know from observation that mere vascularity of a very small surface, without a particle of lymph, may produce faint rubbing noise;*

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* Case of F. Parker, U. C. H. (Males), vol. iv. p. 177, Dec. 1848. A rub may attend the impulse at the apex with knocking sound, independently of any pericardial irritation, as far as can be even suspected; *e. g.* cases of Sus. Roberts and Bonsey, U. C. H., Oct. 1850.

but exudation-matter is its common physical element. In the great majority of cases, exudation-matter forms on both serous surfaces. Dr. Taylor relates a case where, the cardiac surface alone being affected, ordinary friction was almost completely absent. Still, however, it appears to have existed here (single and systolic) to a slight amount about the base. If exudation exist on the posterior aspect of the sac only, friction will commonly be inaudible.

A former attack of pericarditis will or will not prevent the development of friction-sound with a new attack, according to the state in which it has left the serous membrane and cavity of the sac. If it have left a state of perfect agglutination behind it, then, unquestionably, friction is impossible; if of loose adhesions, new lymph may be thrown out between these, and friction will be developed. But, probably, old pericarditis will always have a tendency to limit the extent and regulate the site of new friction-sound.

Calcification of the pericardium (or of its exudation-matter rather), tuberculous and carcinomatous disease may be conceived to produce permanent friction-sound; but I have not observed this. Whether fibrinous, cancerous, or other matter within, and in the walls of, the heart, may, by simply elevating the cardiac surface, produce friction-sound, independently of pericardial irritation, I do not know from experience.

The distinction of pericardial from neighbouring pleural friction, turns mainly upon its difference of rhythm. But sometimes cardiac action produces friction in an inflamed pleura adjoining, the pericardium being unaffected. The distinction of this kind of friction is not always easy. The following circumstances argue in favour of friction of cardiac rhythm being of pleural origin: the limitation of the sound to the edge of the cardiac region; fixity in one or more particular spots; if, when the breath is held, every beat of the heart is not accompanied with friction; steadiness in intensity and quality of the friction-sound. Local dry pleurisy, close to both sides of the heart, sometimes *precedes* dry pericarditis: the distinction of the friction-sounds is then most difficult.*

Pericardial murmur is, in the great majority of cases, easily distinguished from that of endocardial origin by the following characters and circumstances: its rubbing quality; its superficial character; its abrupt limitation and non-transmission in the course of endocardial murmur; its changeableness in precise seat and intensity from hour to hour; the increase it undergoes in sharpness and extent when the patient bends forward; its occasional increase by regulated pressure of the solid stethoscope; its want of perfect synchronism with, or fixed relationship to, the heart's sounds; and its being sometimes accompanied by friction-fremitus, which can scarcely be confounded with valvular thrill. Pericardial murmurs are commonly rougher than *acute* endocardial murmurs. Hope held that a diastolic *rough* sound is of necessity pericardial, endocardial diastolic murmurs never possessing this character; but in this he was absolutely wrong: an aortic diastolic murmur may be very rough. But cases occur where it is next to, or absolutely, impossible to decide on the peri- or endocardial origin of a murmur. This difficulty is oftenest felt about the base and great vessels, and when the sound is of clicking character. Sounds really produced at the valves of the aorta, may not be transmitted along this vessel—as a consequence simply of their feebleness.

The action of the heart sometimes produces rhonchal sounds in the adjacent lung,—cavernous, mucous, subcrepitant, &c. The rhythm of the rhonchi, and their persistence during suspension of the breath, disclose their mechanism.

A double rasping friction-sound, so loud as to be audible at two inches' distance from the chest, existed in Dr. Swett's remarkable case of enlarged granular liver, where the pericardium and valves were perfectly natural (vide p. 177.)

(D.) *Respiratory Murmurs in the cardiac region.*—In the natural state the respiration is more audible over the entire cardiac region, though somewhat enfeebled about the centre. Enlargement of the heart widens the superficial extent of this feebleness; pericardial effusion does so also, and still more in proportion to the percussion-dulness existing,—to so sensible an amount, indeed, that the excess is clinically useful *as an aid* in distinguishing these two states of disease under difficult circumstances.

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* Two cases of this kind (Hayes and F. Smith) recently occurred at the same time: U. C. H., Ward 4, Oct. 1850. Vide Clin. Lect., loc. cit. p. 389.

Pericardial adhesions, especially if attended with adhesion of the pericardium to the ribs, and agglutination of the adjoining pleural surfaces, will maintain audible respiration in the pericardial region, through the course of a subsequent attack of pericarditis with liquid effusion. Under these circumstances, persistence of respiration in front of the heart would, as I have elsewhere shown, be a sign of pericardial adhesions.

(E.) *Vocal resonance in the cardiac region.*—In the state of health, vocal resonance is weak, or actually null, in the præcordial region. In some cases of pericardial effusion, the resonance is not only intensified on the confines of the liquid, but acquires an ægophonic quality. In the only instance in which I have actually observed this, the state of the lung adjoining the distended sac accounted for intensification of resonance,—the fluid simply gave this an ægophonic quality.*

GREAT ARTERIES.

SECTION I.—INSPECTION.

The outline and movements of the large arterial trunks are not visible to the eye in the state of perfect health. In very emaciated persons the pulsation of the aorta in the epigastrium may be seen, however; and, if the arch of that vessel lies

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[*] D. Harley, Consumption Hospital Case Books (Males), vol. i. p. 76.

unusually high, slight impulsive motion may be observable in thin people above the sternal notch. The carotid pulses can scarcely be seen, if the neck be tolerably provided with soft parts: the same statement applies to the smaller arteries. No expansible character can be detected in any of these motions.

With these exceptions, notable visibleness of arterial pulsation, with locomotion, is an unnatural condition, depending on (*a*) general or local excitement of the circulation; (*b*) special disease of the heart; or (*c*) disease of the visible vessel itself.

(*a*) General excitement of the circulation, it is well known, produces visible throbbing action in the innominate artery and carotids, which passes away with that excitement. The vessels, however, simply pulsate, and undergo no locomotion. Visible pulsation of the epigastric aorta may be similarly induced. The arteries leading to an inflamed part often beat visibly.

(*b*) Aortic regurgitant disease, as first shown by Dr. Corrigan, renders the pulsation of the superficial vessels visible: the vessel moves forward too, in the direction of its axis,—sometimes vermicularly. The radial arteries at the wrist, the temporals, and the posterior tibials behind the malleoli are the usual seats of the appearance; but in highly marked cases the carotid, brachial, femoral, and external iliac arteries distinctly present it. To what extent may this sign be trusted as evidence of aortic regurgitation? In the first place, no well-marked case of that disease has ever fallen under my notice in which visibleness in the superficial pulses was not more or less obviously present. In the second place, I have never observed *highly marked and extensive* visible pulsation without aortic regurgitant disease. But in the third place, in aged persons of thin frame, whose vessels are calcified and tortuous, a slight degree of the phenomenon may certainly be noticed in the smaller arteries, independently of any affection of the aortic valves; and if the left ventricle be hypertrophous in such persons, the amount of visibleness will be materially greater; but, still, will fail to affect the larger trunks. I have never known simple or eccentric hypertrophy alone produce this sign in persons under the age of forty, whose limbs were even moderately well covered with flesh: it is affirmed that the peculiarity has occasionally been traced to this cause solely, but the qualification regarding age, my own observation leads me to make, is not referred to. Hope thought that where there was mitral regurgitation or contraction to any amount, aortic regurgitation failed to render the pulse visible. This seems, merely, theoretical, and is positively opposed to what I have seen.

(*c*) The impulse of arteries locally diseased is much better studied by palpation than by the sight.

SECTION II.—APPLICATION OF THE HAND.

The systole of the ventricles conveys to the entire extent of the arterial system an undulatory and somewhat expansible motion (easily felt in vessels, of a certain size, lying within reach of the fingers) known as the arterial pulse or diastole. Each arterial diastole is followed by a contraction or systole. In vessels close to the heart the pulse is perfectly synchronous with the ventricular systole, but falls more and more behind it, in point of time, the further the artery from the central organ. With this qualification, the synchronism of the pulse and ventricular systole is perfect in health; the altered rhythm of the latter, produced by changes of posture (sitting, lying, standing, &c.), and all other physiological causes, is impressed on the former. In diseases of the heart, various provisions of this synchronism occur, and have been already described.

The characters of the pulse which may have diagnostic signification in cardiac disease are as follow:—the diastole may be quick or slow, short or prolonged, soft or hard, loose or tense, empty or full, small or large, equal or unequal in force, and other characters, in successive beats; and the rhythm may vary in the different forms elsewhere noted (p. 172). The stroke may be vibratory, jerking, undulatory, bounding, or reduplicate (bis-feriens): these terms are in themselves sufficiently explanatory of the states to which they refer.

The pulsation of the thoracic aorta can be felt only in one spot in health,—above the sternal notch. If the finger be pressed downwards in that position, the patient's head being at the same time bent forwards, slight impulse is, in the majority of cases, detected: where the arch lies high, there will, of course, be very distinct movement. True aortic impulse is felt directly in the middle line; impulse inclined to the right side originates in the innominate artery. The pulsations of the abdominal aorta may be felt, if pressure proportional to the thickness of the parietes, &c., be made with the hand or stethoscope. The movement is simply one of elevation of variable force. Except in very thin persons, it is impossible to feel the beat of the vessel laterally.

In the various forms of dilatation of the arteries, the dilated portion (unless it be more or less completely filled with solidified fibrine) pulsates with undue force. The character of the movement becomes *hammering*,—its force sufficient, in some cases, to shake the entire trunk and limbs. The pulsation is expansile, as well as heaving, in character; but the distinction of the two movements is not always practically easy. In the case of the thoracic aorta, there is no artifice by which obscure expansile movement may be rendered distinct. In that of the abdominal portion of the vessel, the hands may be sometimes slipped deeply on either side of the vessel, and a double sideward impulsion sometimes detected, which escapes detection when the examination is made in front only. But it must be confessed that the pulsation of the aorta, when pushed forwards by an enlarged vertebra or tumour, cannot always be distinguished by this plan from that of enlargement of the vessel itself.

Arterial pulsation is in various forms of disease accompanied with thrill, occasionally more intense than the valvular variety. Simple dilatation of a vessel, such as the arch of the aorta, especially if it be roughly calcified, is a more efficient cause, than aneurism, of this phenomenon; and a spanæmic state of the blood contributes greatly to intensify it, when other conditions are favourable. Thrill may, in such a combination of circumstances, be felt two or three inches beyond the limits of the dilated vessel. Certain morbid states of the blood, especially spanæmia, will suffice, independently of any organic disease, to produce arterial thrill, which, though slighter in amount, is generally more diffused than that of organic origin.

SECTION III.—PERCUSSION.

In the natural state, the thoracic aorta has no influence on the percussion-sound of the surface beneath which it lies. Even where the arch lies high, the resonance at the sternal notch can scarcely be said, practically speaking, to be affected.

But when the vessel is notably dilated, dulness under percussion, of course, occurs, proportional in extent and intensity to the amount of enlargement,—modified in some degree by the fluid or solid state of its contents, and, to a serious degree, by the vicinity of the enlargement to the surface, and the condition of the intervening textures.

The dulness of a saccular dilatation of a vessel is not coextensive with its dimensions. From its more or less globular form, a limited portion only of the sac reaches the chest-walls, and the observer cannot safely percuss with sufficient force to detect its deeply-seated parts. Hence, practically, an intrathoracic, and even an abdominal, aneurismal sac, is always larger than the results of percussion would indicate. In non-saccular dilatation of the aorta, percussion is, for obvious reasons, a surer guide to the size of the enlargement.

The resistance of a sac filled with fibrine is highly marked; and the deficiency of elasticity so peculiar, as to help in distinguishing the dulness under percussion from that of other states; as, for instance, tuberculisation of adjoining lung. The line of union of simply condensed lung and a saccular dilatation is, however, with much difficulty to be drawn, even with the help of this peculiarity.

It is difficult to fix the smallest amount of dilatation of the arch of the aorta that can be clinically demonstrated. Much will depend on the pains taken in the investigation,—much on the thinness of the chest-walls and the healthiness of other parts; but much more on the precise site of the dilatation. In a case where the dilatation increased the width of the vessel, when cut open, by two inches, and hence increased the diameter of the unopened vessel by only about two thirds of an inch, the fact of its existence, suspected from thrill and undue impulse, was *proved* by percussion. But here the right angle of the arch, the point where the vessel nears the surface most closely, was affected. Such an amount of dilatation could not have been detected in any other part of the arch.* Physical evidence will always be difficult to obtain, where a small sac is situated at the posterior surface of the vessel; but by careful percussion in the course of the arch, a sac as large as a good-sized walnut may be discovered, if it lie anywhere between the second right interspace and the left border of the sternum.

SECTION IV.—AUSCULTATION.

Two sounds, synchronous with each systole and diastole of the heart, are, in the state of health, clearly audible in the course of the pulmonary artery and arch of the aorta: gradually weakening in force in the thoracic division of the latter vessel, they are with difficulty caught in the lumbar region, but by depressing the abdomen with the stethoscope, may readily be heard in front. In some persons, however, a single sound, synchronous with the diastole of the vessel, can alone be detected below the chest.

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* H. Gordelier, U. C. H., Males, vol. iii. p. 331, and Consumption Hospital, vol. i. p. 56;—the patient was extremely thin, too; an accidental aid of no mean importance.

In the carotid and subclavian arteries almost always, in the axillary and femoral in a fair proportion of cases, the sound continues double; in vessels more remote from the heart, a single sound only, synchronous with the arterial diastole, can be heard. The more active the circulation, the larger the vessel, the thicker its walls (provided their elasticity be not impaired), the more marked is the sound perceived on auscultation. It is not unusual to find a single (very rarely a double) sound even in the brachial and radial arteries, where these conditions are favourable. In the vicinity of the heart, the arterial sounds closely resemble the cardiac, in quality, pitch, and proportional duration; at a short distance from the heart, both become equalised in length and loudness; sometimes in the carotids the second is the longer and louder of the two.

If the vessels are full, and the blood of natural composition, the arterial sound is duller, but more prolonged, than when the mass of blood is small, and its quality thin. The arterial sounds are louder in females, children, and thin persons, than in males, adults, and stout people. Their intensity is increased by slight pressure; a blowing murmur takes their place, in a large proportion of persons, under stronger pressure.

The arterial sounds heard in the vicinity of the heart are, doubtless, in great part transmitted from that organ: as the second can rarely be heard at any distance from the heart, the inference indeed arises that it is solely a transmission sound, and that the systole of the arteries (at least of the minor ones) is noiseless. But that the impulsion and friction of the blood against the vessels, and vibrations of their walls, during their diastole, with the current-like motion given to their column of blood by the form of the vessels, generates sound, cannot be doubted; it is not conceivable that sound, audible in the popliteal or radial artery, is the mere result of conduction. Besides, it is not very uncommon to find the first sound in the carotid artery (oftener the right than the left) stronger than at the aortic orifice.

The arteries, like the heart, become the seat of murmurs, either from organic change or independently of this.

(*a*) Organic murmur in the intra-thoracic arteries varies in intensity from a scarcely perceptible sound to one audible even at a slight distance from the surface; is limited to a small portion, or (more rarely) extends over a considerable tract of the vessel; presents all the varieties of quality noted in cardiac murmurs; varying in pitch from that of the word *awe* whispered in inspiration, to a high whistling note; is of distinctly intermittent rhythm, single (systolic or diastolic) or double; and either short and abrupt, or prolonged and slow.

The organic conditions of arterial murmur are referrible to (1) change of form of the vessel; (2) to a modified condition of its internal surface; (3) to both these states combined; and (4) to communication between an artery and some portion of the venous system.

(1) General dilatation, or simple lateral sacculation of an artery, especially if abrupt and well-marked, by altering the direction of the blood current, generates murmur, even if the internal surface be perfectly smooth. Narrowing of a vessel produces a similar effect, by increasing the friction of the blood against the walls at the constricted point.* Murmur of both these kinds is always synchronous with the pulse, and not rough, unless the blood be spanæmic, and the inner walls deficient in smoothness.

(2) Mere atheroma, unless accumulated in unusually large quantity, does not produce murmur; infiltration with induration-matter, by puckering the inner surface irregularly, roughens the sound; erosions of the lining membrane do so more effectually; and calcification of the vessel, if it cause irregular elevations of that membrane, is still more efficient. It seems almost certain that murmur must be produced by particles of exudation-matter studding the surface of an artery recently inflamed; but I have

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* Dr. Corrigan, however, argues with great ingenuity that it is not at the constricted point of a vessel, but in the loose walls beyond, that murmur is produced.

not actually observed the fact. This variety of murmur is always synchronous with the pulse.

(3) In the varieties of aneurism with diseased coats, form and surface are changed so as to produce murmur, which may be systolic, diastolic, or both, and attended or not with sound also. The conditions regulating the time and accompaniments of this murmur, will be described under the head of Aneurism.

(4) Intra-thoracic varicose aneurism, in all its forms, is attended with murmur synchronous with the arterial diastole, sometimes prolonged through its systole.

A certain amount of force in the heart's action is essential to the generation of arterial murmur; increase of that force will convert a soft into a harsh quality instantaneously.

Murmurs heard in the thoracic aorta, single or double, are often merely conducted from the heart. But if a murmur, audible at any part of the arch, be of different pitch, of greater intensity, and of harsher quality than a synchronous murmur at the aortic base, it may be inferred that a cause of intensification exists in the vessel itself. The only source of fallacy would be the chance coexistence of badly conducting materials over the base of the heart, and of excessively good ones over the arch of the aorta. The characters of an arterial murmur will sometimes guide the observer partially to a knowledge of its anatomical cause; but the actual determination of this will mainly turn on the state of other physical signs.

(*b*) Inorganic arterial murmur is commonly softly blowing, if the vessel be ausculted without pressure. If pressure be used, it rises in pitch, and becomes sharply whiffing or whipping (resembling the sound produced by a quick stroke of a riding-whip through the air). It is intermittent, never double, never synchronous with the systole of the vessel, and affects the arterial system extensively, instead of being purely local, as the organic variety. Midway between the organic and inorganic varieties, stands the murmur of an artery, sound in itself, but pressed upon by an adjacent tumor.

The clinical conditions of inorganic arterial murmur, are certain of those of cardiac murmur of the blood-class, especially spanæmia. It is said that plethora produces it;—a statement I have been unable to verify clinically.

VENOUS SYSTEM.

SECTION I.—INSPECTION.

Considerable distension of any particular portion of the venous system indicates the existence of obstruction in the connected main trunk, or in the right side of the heart itself.* Hence an easy clue to the seat of intra-thoracic tumors.

The internal and external jugular veins are the veins most frequently found enlarged,—the right more commonly than the left, when one side only is affected. This obstructive distension, uniform or varicose, even if increasing the size of the external jugular almost to that of the little finger, is unattended either with change in the integuments, hardness or cordiness of the vein, or tenderness under pressure. The common causes of this condition are tricuspid regurgitation, and pressure on the superior cava or innominate veins by intra-thoracic tumor or aneurism; more rarely, simple dilatation of the right cavities of the heart.

The external jugular vein (oftener the right than the left) is occasionally the seat of visible pulsation, especially at its lower part near the clavicle. Irregular in amount and in rhythm (though obviously connected in the main with the ventricular systole), jugular pulsation wants the distinctness of an arterial pulse, and is rather an unsteady intermittent tremulousness than a series of well-defined beats. The effect of inspiration and expiration on the blood in these veins partly explains the irregular rhythm; which may also, in part, be traced to the influence of the auricular systole. The impulse producing it comes visibly from below; and when the vein is emptied by pressure from the clavicle in an upward direction, it re-fills immediately from below, while the pressure is sustained above.

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* It is not intended here to refer to the signs of local diseases of the veins, such as phlebitis, &c.

Lancisi, the original observer of this phenomenon, supposed that it was produced by eccentric hypertrophy of the right ventricle. Hope, holding to this view, explains the impulse by the "impetuous recoil of the tricuspid valve," which repels the blood about to descend into the ventricle with such force that its impulse is propagated back to the jugular veins. Many persons maintain that jugular pulsation only occurs where the tricuspid orifice is too much dilated to admit of closure by its valve, whence ensues regurgitation in the veins during the ventricular systole. Dr. Parkes teaches that, in addition to tricuspid insufficiency, rupture of the valves at the junction of the internal jugular and subclavian veins is a necessary condition of the phenomenon.

I know of no facts positively showing the necessity of such rupture of valves; the vessels may be sufficiently distended to render their valves incompetent, which is all that is required.* The valves, too, may be congenitally absent. Further observation on these points is, however, desirable. But, as concerns the tricuspid orifice? Unquestionably jugular pulsation is most frequently met with in cases of tricuspid insufficiency (though by no means in all of the class); while, as I have decidedly observed it, where the valve was not demonstrably incompetent, in cases of dilated and hypertrophous right ventricle, I cannot refuse to admit that this condition alone may produce it.† If the ventricle be hypertrophous, and the valve insufficient, the pulsation reaches its maximum. It is to be remembered, too, that respiration affects jugular pulsation, emptying the vein in inspiration, distending it in expiration. The parts played severally by respiration and heart-action, may be distinguished by causing the patient to suspend his breath for a moment.

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* *e. g.* Case of Thos. Denham, U. C. H. Feb. 1851, Males, vol. vi. p. 69. Here notable pulsation, both of the jugular and innominate veins, had existed during life; the valve was perfect, but enlargement of the calibre of the veins had obviously rendered it incompetent.

† It is too constantly assumed by observers, that where the tricuspid valve is insufficient to close the orifice at death, it has been so during life also. What proof have we that an instinctive constriction of the orifice does not accommodate the width of the opening to the capabilities of the valve?

The right mammary veins under similar circumstances may be knotty and pulsatile, but I have not seen this without disease of the tricuspid valve.

But veins, much more distant from the chest than these,—the veins, for instance, of the *dorsa of the hands and feet,*—may be the seat of pulsations either of cardiac or of respiratory rhythm, or of both combined. Dr. Jenner has very kindly favoured me with the particulars of three cases illustrating these various rhythms. When the rhythm is cardiac, pressure on one of the pulsatory veins on the back of the hand, increases the strength and distinctness of the pulsations (equal in number to the radial pulse) to the distal side of the point pressed on, annuls them to its proximal side; the respiratory movements exercise no influence on the pulsations. When the rhythm is respiratory, the vein collapses in inspiration rapidly, swells in expiration slowly, and, when pressed on, the pulsation ceases to the distal, increases to the proximal, side of the point pressed on. One of Dr. Jenner's cases exhibits the co-existence of the two sorts of pulsation (respiratory,—and cardiac, by *vis à tergo* through the capillaries, probably) in an infant aged eighteen months, cut off with pneumonia secondary to tubercles. The reason why respiration and cardiac action should exercise this influence on distant veins in some cases of disturbed thoracic action, and not in others, apparently similar, is yet to be discovered.

SECTION II.—APPLICATION OF THE HAND.

Thickening of the walls of the jugular veins sometimes arises in cases of tricuspid regurgitation of long standing. If in such a case those vessels pulsate, it is very probable their diastole will be perceptible to the fingers; but I do not remember ever to have actually observed this.

Visible pulsation is occasionally attended with soft thrill,—a minor degree of the arterial phenomenon of the same name.

SECTION III.—AUSCULTATION.

The venous system, as was originally and most ingeniously shown by Dr. Ogier Ward, is the occasional seat of audible murmurs, which possess one invariable character—that of *continuousness.* Venous murmurs are instantaneously silenced by interrupting the circulation in the veins generating them.

In point of quality, venous murmurs are referrible to four types: the blowing, the whistling, the humming, and the modulated. The blowing varieties may be as soft as the respiration-sounds in health, strongly blowing, loudly blowing (as the sound heard on applying a shell to the ear), or actually roaring. Or, the sound may be cooing or whistling. To the humming type belong various murmurs resembling more or less closely the noise of a humming-top, the buzzing of a fly, the singing of a tea-kettle, &c. Lastly, venous murmurs are sometimes distinctly modulated, consisting of a series of separate tones, capable of musical notation, recurring at tolerably regular intervals, and accompanied by a low hum, which gives the continuous character to the whole.

Inclining rather to softness than roughness, and of moderate intensity (inaudible unless the ear or stethoscope be applied directly to the surface), generally of low pitch, as the word *who*, (when modulated, of course, this is variable,) venous murmur is liable to change in intensity and quality from one moment to another. This change sometimes occurs from some intrinsic untraceable agency; more frequently from some one of the following causes. Acceleration of the circulation intensifies venous murmur; and as inspiration favours the rapid flow of blood in the veins adjoining the thorax, in these veins, at least, that act ought to, and does actually, increase the loudness of an existing murmur. But, on the other hand, suspension of the breath at first exercises even more markedly the same effect; the sharp collision of the blood disks *inter se*, and against the walls in the struggle to move onwards, probably explains this. If the breath be held for any time, the murmur disappears. Any posture which stretches moderately the vein under examination, intensifies its murmurs: if the part be a muscular one,—the thigh, for example,—there is a source of fallacy in the rumbling sound of muscular contraction, which must be guarded against by examination in a relaxed posture also. In the neck, murmur is stronger in the erect than in the lying posture; probably from the greater rapidity of flow in the former. Venous murmur attains its maximum under a certain amount of pressure, ascertainable in each instance only by actual experiment. Less or more pressure weakens and finally obliterates all audible sound. Sex exercises no influence on the intensity of venous murmur, nor, directly, on its frequency. No doubt, it is greatly more commonly observed (perhaps five or six times so) in females than in males; but this depends simply on the disproportionate frequency of its physical conditions in the two sexes. No evidence has ever been adduced, showing that a given state, which fails to generate murmur in a male, will succeed in the case of a female.

The veins in which murmur occurs may, as far as I have observed, be arranged as follows in order of frequency. The external and internal jugulars, on both sides, or on one side only, in the latter case most frequently the right; the subclavian veins; the femoral (I have never failed to find it in these vessels when well developed in the neck, and it may be caught in them sometimes when inaudible in the jugulars); the axillary; the superior cava and innominate veins; the veins of the bend of the elbow; certain abdominal veins;* the pulmonary veins; and the supe-

* Case of Sus. Roberts, U. C. H., Oct. 1850. Continuous hum, coupled

rior longitudinal sinus, especially at its termination in the torcular Herophili.*

Invariably continuous in rhythm, murmur in a vein may be simply continuous, that is, of equable force constantly; or it may be remittently continuous, undergoing intensification and weakening at regular intervals. The type is humming when the rhythm is thus remittent. Two causes of this remittent character have been suggested: the pulsations of an adjoining artery against the sonorous vessel, which give at regular intervals a momentary impetus to the current in the interior of the vein; and the coexistence of ordinary intermittent blowing murmur in the accompanying artery. The majority of instances are fairly explicable on one or other of these principles; some, which are not so, may, it would appear, be explained by intrinsic inequality of force of current in the veins,—an inequality which, we know, positively exists in cases of venous pulse.

Venous murmur may be accompanied or not with inorganic arterial or cardiac (systolic and basic) murmur.

Venous murmurs are so intimately connected clinically with a spanæmic state of the blood, that they constitute its most positive sign; why that state of the blood should engender them, is a mystery. Physically, the vessels are imperfectly filled, loose and vibratile, the blood is thin, and the friction attending its movement (according to a law of Poisseuille's) thereby proportionally increased,—one element of sonorousness. So, too, external pressure, or muscular action, intensifies the sound by similarly affecting the friction of the current; though, if the condition of the blood be highly favourable, no pressure is required, especially when the arrangement of the vessels is such (*e.g.* in the torcular Herophili) as to promote forcible collision of

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with arterial intermittent murmur, a little above and to the *left* of the umbilicus. Here, too, appear cases of continuous hum, audible on deep pressure at the right edge of *some* enlarged spleens.

* Davis, U. C. H. Females, vol. iv. p. 138. In this case a continuous remittent murmur was also audible at both sides of the mid-dorsal spine.

currents arriving from different directions at a conflux. M. Andral has attempted to establish the exact relationship between the amount of spanæmic change and the constancy of venous murmur as follows: if the red corpuscles fall below 80 per 1000, murmur is constant; if they range between 80 and 100, pretty frequent; if between 100 and 115, occasional; if between 115 and 126, murmur is sometimes heard;—never if they reach the average of health.

There are some facts difficult to reconcile with the ordinary notions in this matter. Thus it is well known that in cases of chlorosis treated with iron, colour returns to the tissues, long before venous murmur disappears. On the other hand, Becquerel and Rodier give analyses of the blood of two chlorotic girls, presenting well-marked venous hum, with a mean proportion of 125·1 per 1000 of red corpuscles; certainly an amount falling within the limits of health. It is affirmed, too, by the London Heart Committee, that murmur may be produced in the veins by pressure in a state of robust health; and I have heard it in women of florid complexion, who, as far as I could ascertain, had never been *symptomatically* anæmic. It is averred by Skoda that hydræmic blood has been drawn from persons perfectly free from venous murmur: it was probably not carefully sought for. There is no proof that mere diminution of the mass of the blood will produce venous hum; such diminution, indeed, never takes place without change in composition. Plethora, especially of that kind in which the proportion of the red disks is raised, is an asserted cause of venous murmur.

In all probability the proportion of white corpuscles may have more to do with the murmur than has been suspected. They are increased in many cases of chlorosis, and (as shown by Remak) augment by the repetition of bleeding; now, their increase must entail great increase of friction and labour in the circulation.

The diagnosis of venous murmurs turns essentially on their continuous character; and is excessively easy, except when accidental circumstances occur to render that character obscure.

This happens sometimes about the base of the heart anteriorly, and between the scapulæ in the back. Pulmonary venous murmurs are partially masked by the cardiac sounds. In addition to its peculiar quality, pitch, inconstancy, ready influence by change of posture, as guides to the venous origin of the murmur, its rhythm in respect of the heart's beat will *sometimes* aid in connecting it with the veins. Thus a diastolic murmur at the base (the signs of *organic* disease at the cardiac orifices being deficient) must be venous, according to my experience;—at least an inorganic cardiac murmur of that site and rhythm has never fallen under my notice.

PART II.

DISEASES OF THE LUNGS, HEART, AND GREAT VESSELS.

CHAPTER I.

THE LUNGS AND APPENDAGES.

NEURALGIA OF THE LUNG.

THE parenchyma of the lung is, not only, under ordinary circumstances, endowed with but slight sensibility, but even in the state of inflammation is very rarely the seat of pain, at least of pain demonstrably referrible to itself. Still, pain has occasionally been noticed in central pneumonia, where the pleura had wholly escaped; and various anomalous, and more or less painful, sensations felt by phthisical patients, deeply within the chest, apparently originate within the pulmonary texture. Certain morbid processes in the parenchyma appear then to irritate the branches of the pulmonary nerves.

The only physical condition I have ever succeeded in connecting with these painful sensations (and this only in cases of phthisis), is jerking rhythm of the respiration. The quality of the murmurs may also probably be roughened by their existence.

These sensations are either greatly relieved or altogether removed by counter-irritation, and emollient and anodyne inhalation. They are not so directly, as might be expected, modified by anodynes taken internally.

PLEURODYNIA.

Rheumatism of the intercostal muscles, accompanied, as it is, with more or less acute pain, generally most marked in the infra-axillary and infra-mammary regions, increased by pressure, by deep inspiration, by coughing, movements of the side, and decumbency upon it, simulates pleurisy in its earliest or dry stage. The physical signs, even, are not dissimilar; the movements of expansion and of elevation are diminished in freedom, and their rhythm becomes jerking; the respiratory murmurs are of intermittent weak type, and jerking rhythm; the percussion-sound is not perceptibly altered. Friction-sound is of little use in the distinction of the two affections,—for the grazing variety, that appertains to dry pleurisy, is often wanting in that disease, and the jerking rhythm of pleurodynic respiration may so closely simulate it, as to leave a cautious observer in doubt. If with pleurodynia there be a chance co-existence of febrile action, cough and slight bronchitis, a positive diagnosis should be refrained from, until a certain number of hours having elapsed, the rubbing friction-sound of plastic exudation, if the case be one of pleurisy, will have established the fact beyond the possibility of doubt.

True rheumatic pleurodynia yields rapidly to cupping, dry cupping, anodyne and stimulant liniments, and the internal use of colchicum and an alkali.

INTERCOSTAL NEURALGIA.

The intercostal nerves, especially on the left side, and from the sixth to the ninth, are not unfrequently the seat of neuralgia, which, in respect of diseases of the lung, derives its interest from the possibility of its being confounded with pleuritic pain, and from its being pretty frequently associated with phthisis. The pain is severe, occurs paroxysmally, follows the course of the affected nerve, and, if there be co-existent palpitation, may (especially if combined with brachio-cephalic neuralgia) simulate, in its paroxysm, an attack of angina pectoris. Generally speaking, three tender points (as was first, I believe, shown by M. Valleix) may be detected by pressure in the course of the nerve,—one in the vertebral groove, another about the axillary region, a third in front towards the terminal ramusculi. In the female, intercostal neuralgia is often associated with that of the mammary gland, and with spinal irritation.

The physical signs are those of pleurodynia; impaired chest-motion, with weak jerking respiration,—the percussion signs being negative. The three painful points in the course of the nerve, point to the true nature of the affection, distinguishing it from periosteitis, and all pains of intra-thoracic origin.

If the tenderness be extreme at any one of the three points, a few leeches are requisite; subsequently flying blisters will, as concerns local measures, often complete the cure. The endermic use of morphia, and inunction with belladonna or aconitina ointments, combined with purgatives and iron and quinine internally, will triumph, generally, over the most obstinate case.

BRONCHITIS.

Inflammation of the mucous membrane of the bronchial tubes, or bronchitis, perhaps the most common of pulmonary diseases, occurs in the acute and chronic forms.

ACUTE BRONCHITIS.

I. Simple primary acute bronchitis of the larger tubes, in the adult, is commonly ushered in by coryza, sore throat, and slight hoarseness, chilliness (scarcely amounting to rigors), with lassitude and continued pains in the limbs, and frequent pulse. The occurrence of coryza is significant of the primary character of the disease,—tuberculous bronchitis very rarely originates with this symptom.

The disease being established, more or less discomfort and pain are felt behind the sternum,—a sensation of heat, soreness, or rawness of the bronchial surfaces, increased, perhaps, to acute pain by coughing, and attended with a sensation of oppressed breathing. The respiration is increased in frequency, slightly out of proportion with the pulse,—in severe cases, notably so. The cough, an essential feature of the disease, at first short and dry or nearly so, occasionally paroxysmal, and severest after sleep, loud, hoarse, and ringing, is attended, after the lapse of one or two days (when it becomes loose), with expectoration of frothy mucus, watery in the main, ropy in some measure, of saline taste, faintly yellowish, yellowish green, or greyish yellow colour, free from blood visible to the naked eye (though blood-disks in small number, in company with epithelium and exudation-corpuscles, may be found with the microscope,) varying greatly in quantity, and gradually becoming muco-purulent. The sputa run together into a single mass, except in rare instances, when they present the nummulated form, with perfect opacity: I have seen this even in children in the bronchitis of measles. Referred, by the patient, sometimes to the sternal region, the cough is more frequently brought on by a tickling feel about the trachea and larynx, where the patient will consequently obstinately contend his whole ailment lies.

Lasting from four or five days to two or three weeks, in cases of complete recovery (which make the majority), attended with febrile action of sthenic or asthenic type, the acute disease may terminate in its chronic form, or actually prove fatal: the latter result only occurs in infants, in aged persons, and in those debilitated, constitutionally, by excesses of various kinds, or by chronic diseases.

But there is a severer form of the disease, where the capillary tubes, as well as those of medium and large calibre, are implicated. Here, though there be no more, or even less, pain than in the previous form, the danger is infinitely greater: the pulse is extremely frequent, and the respiration disproportionately hurried; painful sense of dyspnœa, livid, leaden, or cyanotic tint of countenance, coolness of the expired air and of the surface of the body, glutinousness and viscidity of the sputa, and difficulty of expectoration, all point to the asphyxiating tendency of the disease—a tendency explained by the amount of bronchial surface involved, and the adhesive character of its secretion. In these cases the vital powers fail rapidly, unless vigorous measures be adopted; and quiet delirium, with slight convulsions, precedes fatal coma. The accidental blocking up of a large bronchus by adhesive secretion occasionally hastens this termination.

Physical signs.—Little of a positive kind is discovered by inspection in slight bronchitis; the perversion of movement, unless there be emphysema present, is not sufficient to attract the eye. If the disease be severe, the costo-abdominal expansion-motions assume the characters of healthy *forced* breathing,—the costal increase in amount, the abdominal decrease. The form of the chest is not perceptibly altered.

The hand, applied to the surface, occasionally detects distinct rhonchal fremitus,—its presence furnishes a rough guide to the seat of the rhonchus, as it will scarcely be transmitted to the surface, if a deep bronchus be the source of vibration. It may be more distinctly perceptible during inspiration than expiration, or *vice versâ*. Dr. Stokes believes that it is more marked in the child and female than in the adult male, and at the middle and inferior parts of the chest than the superior. I have found it very remarkably developed in infants of from six to twelve months old. The state of vocal fremitus varies; it sometimes exceeds the average of health.

The percussion-sound, as a rule, is not impaired in clearness, and may be even a little raised above the pitch of health. The limits of clearness extend in front a little further downwards than natural, and expiration has less effect than in health in diminishing the area and amount of pulmonary resonance. The lung is held to a certain extent in a state of mechanical distension, from diminished elasticity of its tubes and substance, and from imprisonment of air by glutinous mucus.

In certain cases, slight dulness under percussion may be detected, especially at the postero-inferior parts of the chest. But this condition of sound in simple bronchitis is singularly rare; and when we reflect that considerable turgescence and thickening of the mucous membrane over a large extent of surface form part of the anatomical features of the disease, the usual clearness of resonance affords fair ground for surprise. The fact of such clearness existing is an important one; as it will enable us to infer the idiopathic character of the disease, and conclude without hesitation that it does not depend upon or attend tuberculous deposition.

The imperfect resonance in exceptional cases is variously produced. Sometimes depending upon accumulation of bronchial secretion, it is then chiefly observed at the base and posteriorly, and occurs more especially in subjects of debilitated constitution, or in those labouring under prostrating diseases,—as for example, continued fever—of which the bronchitis is only a secondary condition. In these cases there is often some congestion (or more rarely œdema) of the lung, which takes its part in producing the deficiency of clearness. Occasionally the dulness seems traceable to collapse of the lung, consequent on pressure on a main bronchus,—the bronchitic secretion contributing its share, at the same time, of defective resonance. (Case of Mary Ransom, U. C. H., Nov., 1848).

Reference has already been made (page 63) to the occasional occurrence of a pseudo cracked-metal resonance in the bronchitis of young children especially. I would add, to what is there said on the subject, that unlike the true cavernous sign, this simulation of it is changeable in place.

By auscultation, we learn that the respiratory murmurs, weakened, sometimes, even to suppression, in the tissue communicating with the affected tubes, are exaggerated on its confines and elsewhere,—hence especially so in the upper parts of the chest. The murmurs, dry and harsh in quality, are accompanied and it may be masked more or less perfectly, by sonorous, sibilant, and mucous rhonchi, in various combinations,—the former often of musical quality. The vocal resonance is not perceptibly affected as a rule; but in some cases, probably from nasal character in the voice, it has a sniffling quality,—which can only be confounded with ægophony, through carelessness, or the influence of preconceived theory.

As a general rule, the sonorous and sibilant rhonchi are most marked and constant in the early or dry stage of bronchitis; the mucous in the second, or that of secretion. But both orders of sound are frequently combined in the second stage; and in some cases secretion occurs so rapidly that mucous rhonchus is audible from the first. When the secretion is very abundant, distinct agitation of the fluid in the tubes may be caused by the action of the heart. As was first observed by Dr. Stokes, each pulsation of the heart then causes a corresponding rhonchal sound, continuing when the breath is held.

In idiopathic capillary bronchitis, in addition to the signs belonging to bronchitis generally, (and it is to be understood that the dry rhonchi existing in the larger tubes may be absent altogether, or nearly so, in this variety of the disease), auscultation discovers true subcrepitant rhonchus at both bases posteriorly; fine mucous rhonchus higher up. If abundant and minute in its bubble , this rhonchus indicates very positively that the capillary tubes are inflamed; but as with the converse characters, it certainly occurs in cases running a mild course, gravitation of fluid from the larger tubes above to the smaller below, is probably sufficient to produce it. If limited to one base, or to one or both apices, the bronchitis it depends on is either of emphysematous or tuberculous origin.

In intense bronchitis of both lungs, especially where any slight emphysema pre-existed, the bulk of the organs may be sufficiently increased, to push the heart slightly downwards, and to the right (the organ will beat mainly at the left costal angle), and depress to a trifling amount the diaphragm and subjacent viscera.

II. The treatment of acute bronchitis in the adult is sufficiently simple. Venesection, not often required in town-practice, is advisable to the extent of twelve or fourteen ounces, where an extensive surface is involved, where the constitution is strong, and the febrile action positively of sthenic type. Rarely is repetition of general blood-letting called for by the violence of the disease; and while the abstraction of large quantities of blood, with the view of putting an immediate close to the disease, is perfectly chimerical, such sacrifice of blood is useless for an object assigned by some writers,—the *prevention* of pneumonia, seeing that in the adult idiopathic inflammation of the tubes does not pass on to the parenchyma. In the ordinary class of cases, cupping between the scapulæ (to six or eight ounces), or the application of from twelve to twenty leeches to the upper-sternal, infra-clavicular, or axillary regions, will suffice in the way of a first blood-letting,—to be repeated, should the relief to the breathing (as shown especially by the number of respirations per minute) be only temporary. Dry cupping will sometimes with propriety be substituted in weakly persons on the second and subsequent occasions.

Although not demonstrably so efficient in bronchitis as in pneumonia, tartarised antimony is the most effectual agent known in controlling acute sthenic bronchitis. In divided doses of from four to ten grains in the twenty-four hours, it appears to hasten resolution very sensibly in the milder cases, and advances the secretive stage in the severer. If the tendency to depression under the influence of the antimony be very marked—marked enough to excite fear that the vital powers may be too much lowered—calomel and opium may be employed instead. I entertain no question as to the superiority of tartarised antimony under ordinary circumstances.

Full secretion from the tubes being established, and the febrile action lowered, the application of a full-sized blister to the sternum, or between the scapulæ (here, in consequence of gravitation, it draws off most fluid), becomes most serviceable;—mustard-poultices may, even from the first, be employed. Diaphoretics and expectorants, containing liquor ammoniæ acetatis, vinum ipecacuanhæ, paregoric, and similar agents, are now advisable,—and these may gradually be replaced by preparations of a more stimulant character, such as tincture of squills, and the ammoniated tincture of opium of the Edinburgh Pharmacopœia. Dilute hydrocyanic acid and the tincture of lobelia inflata, especially if there be spasmodic tendency in the cough, are useful adjuncts. In the advanced stage of the disease, especially if there be inclination to lapse into the chronic form, carbonate of ammonia, senega, the balsamic medicines, copaiba, gum-ammoniacum, &c., are distinctly serviceable in moderating the amount of secretion, facilitating its discharge, and so relieving dyspnœa.

Throughout the whole course of the disease, the bowels should be kept freely open (I have never seen any utility in counter-irritant purgation, however); it would be difficult to prove, it is true, that the duration of the disease is prolonged by confinement of the bowels, but most certainly fulness of the abdomen increases dyspnæa, and discomfort in the chest.

The temperature of the room should range from 63° to 66° Fahrenheit (or even higher under special circumstances, such as previous residence of the patient in a warm climate); the moisture of the atmosphere may be regulated according to the patient's feelings by evaporating water from a dish near the bed (I have known violent paroxysms of cough and dyspnæa relieved by this simple plan); and occasional ventilation, the patient's head being covered at the time, is essential to his early recovery,—he wants all obtainable oxygen. Flannel should be put on next the skin, if not previously worn.

In the severer asphyxiating form of the disease, common among the aged, depletory measures must be employed with the extremest caution. The abstraction of a few ounces of blood is sometimes followed by vital depression, which very manifestly hastens the fatal issue, and is perhaps occasionally its real cause. Dry cupping, mustard poultices and blisters to the chest, along with stimulant expectorants, especially the sesqui-carbonate of ammonia in doses of from three to six grains every third or fourth hour, constitute the staple of the treatment.

CHRONIC BRONCHITIS.

I. Chronic bronchitis, like the acute disease, varies greatly in severity. In one class of cases slight cough, with moderate and easy muco-purulent expectoration, and with little or no post-sternal soreness or pain, affecting but very slightly the general health, appetite, and flesh, appearing in winter, and ceasing on the approach of the mild season, constitutes the whole of the disease: this is the slightest form of "winter cough." In a second class of cases the cough is more violent, and more constant, severest in the mornings; the expectoration scanty and adhesive, or easy and copious,—in the latter case consisting of large nummulated masses, floating, semi-sinking or sinking in water, non-aerated or scarcely aerated, yellowish-green, deep green, or in very rare cases of a tint almost like Scheele's green, remaining separate, or forming a single liquid collection, slightly streaked occasionally with blood, especially if the left heart be obstructed in the slightest degree, but never accompanied with actual hæmoptysis,* of peculiar nauseous odour, sometimes fœtid, smelling like wet plaster, or putrescent matter. There is no great pain, heat, or soreness in the chest, except after fits of coughing, when it is mainly post-sternal. The respiration ranges scarcely out of proportion with the pulse in frequency,—both being raised, slightly (as the habitual state), materially (during and shortly after paroxysms of coughing), above the individual standard of health. The appetite fails, the sleep becomes broken, and flesh wastes very perceptibly: I have known as much weight lost during the first three weeks of an annual recurrence of chronic bronchitis, as during the same period in the average of cases of consumption in active progress;—but in bronchitis the weight ceases to diminish after a certain time; in phthisis its diminution holds on.

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* "Expectoration of blood, in persons labouring under chronic bronchitis, with or without emphysema, but without notable disease of the heart, justifies in itself a suspicion of the existence of latent tubercles."—Author's Report on Phthisis, as observed at the Brompton Hospital; Brit. and For. Med. Chir. Rev., January, 1849.

If an acute attack or severe exacerbation of the disease occur in a person afflicted with this serious variety of the chronic form, the expectoration becomes in part more viscid, transparent, and highly frothy for a variable number of hours or days; then relapsing into the simply purulent state, grows so excessively abundant as in itself to account partly for the rapid debility that ensues. The laboured respiration (hurried somewhat out of proportion to the pulse), lividity of the lips, malar bones, chin, tip of the nose and finger-ends, coolness of the hands, feet, and, it may be, of the extremities generally, clammy perspiration, sometimes rather copious, coolness of the expired air, inability to lie down, sensations of oppression and want of air, all point to the asphyxiating character of the attack. Fitful dozes give but temporary relief, and, leading to accumulation of secretion, increase the suffering felt on waking. Here is a condition of extreme danger, principally observed in the aged (*senile* bronchitis) and one of the most frequent causes of their death.

In a third class of cases, the prominent feature of the disease is a peculiar flux from the bronchi,—whence the name *bronchorrhœa.* In these cases paroxysms of cough and dyspnœa, which may be of almost daily occurrence, or even more frequent, are relieved by copious expectoration of a thin watery fluid, or of a ropy, gluey, transparent substance, like raw white of egg mixed with water (a quarter of a pint of this may be secreted in the course of half an hour on the decline of a paroxysm). Though sometimes fatal to old people, from their want of power to throw off the accumulated discharge, this form of the disease seems occasionally useful, when slight, in relieving pulmonary congestion dependent on mitral disease, and should not under these circumstances, without mature consideration, be removed—at least completely.

There is a fourth variety of chronic bronchitis, to which the rather contradictory name of *dry catarrh* was given by Laennec, characterised by exceedingly troublesome cough, oppression of breathing, tightness of the chest, and sometimes extreme dyspnœa;—expectoration being totally deficient, or consisting of semi-transparent, small, grey, pearl-like, roundish particles. An attack of ordinary bronchitis, with muco-purulent secretion, may occur in such cases, and put a period for a time to the chronic disease. This affection is clinically very closely allied to emphysema.

The physical signs of chronic bronchitis are in the main those of the acute disease. There are certain modifications worth attention, however, especially as no single specific sign distinctive of the two stages exists. The percussion-sound at the posterior bases, in cases of chronic bronchitis with acute recrudescence, may become markedly dull; and the respiration acquire a bronchial or even diffused blowing quality. I have known this state mistaken both for pneumonia and for pleuritic effusion. When, as sometimes happens, one side only is thus impaired in resonance, the error is very easily committed, and probably furnishes a clue to the alleged enormous frequency of fatal pneumonia among the aged in some localities. The persistence of vocal fremitus will distinguish the dulness of bronchitis from that of effusion; the deficiency of true tubular metallic breathing from that of pneumonia. In the latter case, too, the pulse-respiration ratio may be appealed to with confidence; it is never perverted, even in this *accumulative* bronchitis, to the degree that it is in pneumonia. The vocal resonance may be strongly bronchophonic, but it is not tubular, sniffling, and metallic.

Chronic bronchitis tends to distend the lungs *generally* (though, from bronchial obstruction in some spots, it may have the reverse effect on them *locally*), and hence to widen the chest, and even depress somewhat the heart and diaphragm. But it is very difficult to demonstrate to what extent bronchitis is capable of producing these changes; it is so constantly associated with their more active and positive cause—emphysema.

In a certain proportion of cases of chronic bronchitis, the structure and form of the tubes undergo serious change;—the longitudinal and circular fibres grow hypertrophous; the walls, generally firm and rigid; the mucous membrane thickens; the cellular coat greatly increases in depth; the cartilages of the larger tubes calcify; while the calibre of one or several tubes widens, in a globular or glove-like form, and the surrounding lung-substance, condensed by pressure, is besides often locally consolidated by chronic pneumonia. This condition, familiarly known as *dilatation of the bronchi*, produces special physical signs, and requires separate consideration.

The bulk of a lung having several of its bronchi dilated, falls below the average, especially in the neighbourhood of the widened tubes. This comes of the co-existing condensation of lung-substance. Hence it is, that instead of any tendency to local bulging, there may be distinct depression of the corresponding chest-surface. And in those cases of dilated bronchi, where the intra-parenchymatous and intra-lobular cellular tissue of the lung is infiltrated extensively with induration-matter (Corrigan's cirrhosis), the affected side generally may be the seat of marked retraction.* Generally speaking, the vocal fremitus is increased in intensity. I have, however, known it certainly not raised above, perhaps slightly diminished below, the natural standard, in a case of extensive and general dilatation of the tubes (in a child aged twelve).

I have never known dilatation of the tubes, or even of a single tube, to any notable extent, unattended *either* with dulness under percussion (commensurate with the extent of attendant condensation), *or* with some form of tubular resonance. In certain cases, where the resonance is alleged to have been "clear," the so-called clearness was doubtless of the latter unnatural quality.

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* *E. g.* Case of Samuel Osmond, U. C. H., Males, vol. iv., p. 341.

The respiratory murmurs are simply harsh, or bronchial, or, more commonly, of diffused blowing type. When a single tube is globularly dilated, and to a considerable size, the respiration may be distinctly cavernous in quality, and, under the same circumstances, the ordinary dry or moist bronchial rhonchi may be superseded by the dry or moist cavernous varieties. The vocal resonance varies: totally deficient in some cases, temporarily, or, to all appearances, persistently; in others it is strongly bronchophonic, and may even assume pectoriloquous quality. The heart's sounds may be transmitted with undue intensity through the seat of dilatation.

Dilatation of the bronchi secondary to pneumonia (acute and chronic), "cirrhosis" of the lung, pleurisy, and pertussis will be considered with those diseases.

The addition of dilatation of the tubes to chronic inflammation of their mucous membrane, seriously increases the gravity of the latter disease. Here it is that the expectoration is most abundant, most opaque and solid, most thoroughly purulent, and hence most wasting to the system. The aeration of the blood is rendered difficult by the altered structure of the mucous membrane; hence lividity of the face commonly exists to a marked amount. The long continuance of the disease tends to produce enlargement and thickening of the right ventricle, but I have never observed hæmoptysis unless where there was co-existent mitral disease or pulmonary tubercle. Some degree of night-sweating occasionally occurs; and the weight of the individual may fall very considerably below the standard of health.

This symptomatic state, far from dissimilar to that of phthisis, may coexist with physical signs, so like those of excavation, that it is next to impossible sometimes to affirm with certainty whether a given case be one of tubercle with cavity, or of globularly dilated bronchus with surrounding induration.* The distinction of the two kinds of cavity may, however, in the majority of cases, be established through the following points. In phthisis, percussion is dull *above* the clavicle; not so in dilated bronchus;—below the clavicle, too, the dulness is greater and more extensive in the former than in the latter case. The signs of tuberculous excavation are found at the apex; those of dilated bronchus generally lower,—say at the union of the upper with the middle third of the chest. When tubercle has reached the excavation-stage, flattening of the surface is habitually more marked under the clavicle than it ever is in bronchial dilatation. I have never known hæmoptysis produced by chronic bronchitis with dilatation alone;—if hæmoptysis exist, and there be no evidence of mitral disease, the inference that the excavation is tuberculous becomes matter of necessity. *Extreme* emaciation does not, as far as I have seen, come of the bronchial disease alone. The course of the physical signs will avail us also, if the case continue for a time under observation. In phthisis the signs are, as a rule, constantly increasing in degree and extent; in bronchial dilatation, they may remain for months unaltered in both these respects: dulness under percussion, as remarked by Dr. Stokes, precedes the signs of cavity in phthisis, and does not occur till after them in bronchial dilatation;—to the latter clause, however, I have seen exceptions.

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* Case lxi. of Louis, Phthisie, 2ème éd., p. 562, is sufficient proof of this.

I once met with a case * where the conditions of bronchial dilatation were sufficiently defined to justify the diagnosis of that state,—and where, after death, both bronchial dilatation and recent growing tuberculous excavation were found. This compound state is, probably, beyond the reach of diagnosis.

II. In the *treatment* of chronic bronchitis (with or without dilatation), it is very rarely advisable to take blood from the arm, even during acute exacerbations;—the strength fails rapidly in such cases from loss of blood; and to bleeding at the outset, rapid asphyxia at the close, from inability to expectorate, may often be traced. A few ounces, say, in ordinary cases, about four or five, taken locally by leeching or cupping, are as much as may be abstracted without fear. Free dry cupping, with flying blisters applied occasionally to different parts of the chest, are among the most effectual means of counteracting acute excerbations. In the purely chronic state, counter-irritation, with tartar emetic, croton oil, or the turpentine and strong acetic acid liniments, is essential in the treatment, unless emaciation be very far advanced, or the skin peculiarly irritable. The inhalation of tar vapour, creasote vapour, iodine, or chlorine, most unquestionably reduces the irritability of the mucous membrane, and the quantity of secretion; the results of M. Cottereau, with chlorine inhalations, are peculiarly important, and show that singular advantage may be obtained through them, even where the general symptoms closely simulate those of phthisis.*

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* Ann Harrison, U. C. H., Jan. 22, 1848.

The choice of expectorant medicines will vary with the condition of the discharge from the tubes. If there be little expectoration, an excited circulation, and a tendency to congestion of the parenchyma, tartar emetic in small doses, ipecacuanha, or colchicum, are the best agents, combined variously with hydrocyanic acid, lobelia inflata, belladonna, stramonium, hyoscyamus, &c. If there be but little vascular excitement, squill, senega, ammoniacum, with opium, are preferable combinations. Balsam copaiba and the compound tincture of benzoin may be joined to the above agents,—the former especially seems to exercise some specific effect on the mucous membrane. Medicinal naphtha may be used to control (which it certainly does) superabundant discharge.

The jellies made from Iceland and Carrageen moss both soothe the cough and afford nourishment. If emaciation occur, cod-liver oil should decidedly be employed;—many of the good effects of the oil, as observed in phthisis, are yet more readily produced in chronic bronchitis. Bark and the mineral acids are useful in controlling debility, and improving the appetite.

The diet should be nutritious and non-stimulant. Where circumstances permit, change of climate should be tried,—the selection of a spot mainly turning on the dry or moist character of the bronchitis. The dry form is benefited by the climates of Torquay, Penzance, Ventnor, Pau, Pisa, Rome, and Madeira; the moist by those of Hastings, Clifton, Nice, Genoa, Naples, Cadiz, and Egypt.

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* Louis, Op. cit. p. 620.

During the paroxysm of *bronchorrhœa*, the hot bath, sinapisms to the extremities, emetics, full doses of lobelia inflata, and, if there be failure of vital power, sesquicarbonate of ammonia, are the chief remedies.

In the treatment of an acute attack, supervening on the chronic disease in a person of advanced years, the caution already given as to blood-letting in simple acute asphyxiating bronchitis, seems to me of yet greater importance. Here it is not the inflammation that kills; it is the vast accumulation of muco-purulent secretion supplied by a congested surface,—secretion which prevents oxygenation of the blood, and which the strength of the patient fails to throw off—that really kills: the brain and tissues become poisoned, too, with venous blood. Sesquicarbonate of ammonia is, in such cases, required almost from the first: it is best given in combination with squill and nitric ether. If any sinking tendency appear, chloric ether acts as a more powerful stimulus in these cases than any medicine I am acquainted with.

It is commonly held that the fatal result is immediately brought about by pneumonia: a notion derived sometimes from the occurrence of dulness under percussion at one or both bases, —sometimes from *post-mortem* examination,—sometimes from both sources. I have already pointed out the source of fallacy in the percussion-dulness referred to; and I have great doubts of certain consolidations found after death (peri-pneumonia *notha* in more senses than one) being truly pneumonic. It has not occurred to me to meet with such consolidation, except where there was co-existent heart-disease, and more especially of the mitral orifice,—consolidation hence obviously mechanically, and not actively, congestive.

In a practical work, it is unnecessary to consider all the varieties of bronchitis; but a certain number of them possess characters so peculiar, that special reference to them seems unavoidable. These are plastic and mechanical bronchitis, hay asthma, influenza, and syphilitic bronchitis.

PLASTIC BRONCHITIS.

Plastic bronchitis, an affection of great rarity, is anatomically characterised by the formation of solid or tubular concretions of exudation-matter of low type * within the bronchial tubes, reaching, more or less extensively, from their finest to their largest divisions. The disease has little tendency to spread upwards: the trachea remains unaffected; the voice, though sometimes becoming slightly husky, habitually retains its natural quality and strength. On the other hand, plastic inflammation extends downwards from the larynx to the bronchi in a small proportion of cases of croup,—but with these cases we have nothing to do here. Clinically the disease is distinguished by its chronicity, and frequent acute recrudescences, and its comparatively slight influence on the general health. The physical signs are also peculiar: disappearance of all respiratory murmurs in given spots of the lung from time to time, marks complete obstruction of the communicating bronchus,—and dulness, as complete as in pneumonic consolidation, probably from collapse of the lung-substance, may occur coextensively with the deficiency of respiration.† Local pneumonia, attended with pain, true crepitant rhonchus, and blowing respiration, also occasionally occurs in these cases,—generally speaking, running its course uninfluenced, at least perceptibly, by ordinary treatment. Where

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* I have found them to contain exudation-cells; some nucleated, the majority not so. In the main, the substance is fibrillar or amorphous.

† *E. g.* Case of Jane Moss, U. C. H., vol. i. p. 187 (1846), and vol. iii. p. 83 (1848). Expectoration of casts of the tubes commenced, in this case, in the spring of 1843, and, with occasional intermission, has continued to the present time (Autumn of 1850).

a very large tube chances to be blocked up, asphyxia may be temporarily threatened; * and oppression of breathing, disproportionate to the apparent amount of disease, is always a prominent symptom.

The expectoration of the casts is generally preceded by some hours' dyspnœa and hacking dry cough; and during the periods of acute attack, I have found the pulse-respiration ratio vary from 2·2 :1 to 3·5 :1. During these attacks, casts of notable size are brought up generally from three to six or seven times a week, —but small fragments are much more frequently expectorated. Unless the sputa be closely examined under water, particles of concretion escape notice amid the viscid mucus with which they are generally associated.

Streaks of blood, either on the external surface of the casts, or, more rarely, on their internal surface, (if they be tubular) are not uncommonly seen; and spitting of florid blood in streaks, or even in drops, mixed with mucus, for a short while after their expectoration, occasionally occurs. This is especially the case at the height of the acute attack. As this wears off, the concretions and the expectoration become bloodless. Cases have been observed, however, in which copious hæmoptysis had occurred for some time previous to the expectoration of solid casts of the tubes. The nature of these cases, however, requires further investigation. It appears very improbable that they belong to the same class as true plastic bronchitis; the concretions are very probably simple fibrinous coagula from hæmoptoic blood, itself the result of tuberculous disease. In no case of the kind that I have read the record of, was the absence of tuberculous disease proved; and, on the other hand, the occurrence of moulded coagula in tuberculous hæmoptysis, though, for obvious reasons, rare, is sometimes (I have seen it myself) positively observed.

During the period of acute seizures the treatment is to be conducted on the same principles as if the secretion-products were of the ordinary kind. I have not seen any benefit derived from

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* U. C. Museum, No. 2124.

[illegible] ?

mercurial action on the system. The young practitioner must not confound the local dulness under percussion, which may come on in a few hours in these cases, and depends on obstruction of tubes, with true pneumonic loss of resonance. The weak or suppressed respiration of the former condition, the tubular blowing of the latter, will distinguish the cases.

Few affections of the lungs are more difficult to cure permanently than this. Theory leads to the use of iodine by inhalation and otherwise, and suggests a prolonged trial of alkalies, as diminishing the tendency to hyperinosis. I have perseveringly employed these remedies without any permanent effect on the disease. It disappears for a time, to return again without obvious cause. There is obviously a *diathesis* to be contended with.

MECHANICAL BRONCHITIS.

Under this head fall those well-marked varieties of bronchitis induced by the inhalation of irritating particles of various kinds. The knife-grinders' rot is primarily mere bronchitis produced by the entry into the tubes of metallic particles and gritty dust from the grinding-stones; miners (whether coal or other), not, as was once erroneously supposed, from the inhalation of coal-dust, but really from that of the soot of the oil-lamps used in working, where the safety-lamp is not employed, are subject to a similar disease; so, too, are quarry-men, cotton-batters, &c. In all these cases the disease (grinders' rot, black phthisis, stone-phthisis, cotton-phthisis, &c.), is essentially bronchitis at the outset, which becomes chronic, is followed by dilatation of the tubes, and, eventually, inflammatory destruction and excavation of the lung-substance itself. But there is no connection between this destruction and the presence of tubercle,—which, if it exist, is purely accidental.

The physical signs are those of bronchitis, dilated bronchi, occasionally of emphysema, of consolidation, and, finally, of excavation. The general symptoms are not proportional in severity to the local disease; whence a distinction between these affections and phthisis. The knowledge of the cause also aids in the diagnosis.

Cure is impossible, unless the patient change his occupation. Various mechanical contrivances have been invented for the prevention of these diseases, especially among knife-grinders. Abraham's magnetic mouthpiece attracts metallic particles, but has no effect on the stone-grit. Dr. C. Holland's revolving fan, acting by a strong current upon the spot where the metallic dust and stone-grit are formed, seems to be more successful.

HAY ASTHMA.

A singular variety of bronchitis, which has been supposed to follow the inhalation of the aroma of the sweet-smelling spring grass* (anthoxanthum odoratum), is known under this name. The entire naso-pulmonary mucous tract is implicated, however; sneezing, irritation in the nostrils, and flux (common coryza, in short), soreness and prickling sensations in the throat, dyspnœa, post-sternal oppression and rawness, cough, and, towards the close of a seizure, thin mucous and watery expectoration, are the symptoms of the affection.

The complaint occurs only at the periods of hay-making, or when the odour of grass is powerful; and is of exceedingly rare occurrence. The susceptibility to these emanations, indeed, constitutes a very remarkable example of unalterable idiosyncracy. Persons who have once suffered, invariably have a return of the disease, if exposed even in a slight degree to the specific cause.

The only effectual way of preventing an attack is by removing at the season to the sea-side,—by getting out of the reach of grass and hay. It is affirmed that a course of sulphate of iron and quinine (Gordon), and the use of the shower-bath, removed the disease in two persons who had been its annual victims for fifteen or twenty years. During the seizure, the æthereal tincture of lobelia inflata is a valuable remedy; there seems, in truth, to be some spasmodic element in the disease. Dr. Elliotson believes he has observed benefit felt from breathing an atmosphere with chlorine diffused through it,—saucers of the chlorides being placed in the rooms of the patient's house, and the face washed with a weak chlorinated solution.

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* Gordon, Medical Gazette, vol. iv.

INFLUENZA.

I. Influenza, or epidemic catarrh, though claiming a place more naturally among bronchitic diseases than any others of the respiratory passages, is evidently, even in its local manifestations, an affection *sui generis*, and of much more extensive seat (even anatomically speaking) than its classification with bronchitis would seem to signify. Still, in the majority of cases, the most prominent local symptoms are those of bronchitis.

Essentially constituted by catarrh, with nervous and muscular prostration, influenza sets in with lassitude, chill (rarely actual rigors), and aching pains in the limbs; and more frequently, perhaps, affects the mucous membrane of the throat at the outset than that of the nose or air-passages. So, too, nausea and vomiting may precede all other symptoms, showing that the surface of the alimentary canal early participates in the disease.

Heat and dryness of skin, frontal headache, sometimes excessively severe, small, weak, and frequent pulse, cough of variable severity, expectoration scanty and pituitous, slight dyspnœa, uneasy pain behind the sternum, tenderness under pressure, and sensation of rawness at the epigastrium, white furred tongue, nausea, absolute anorexia, occasionally vomiting, diarrhœa (trifling in amount), vertigo, tinnitus aurium, pains in the neck, scalp, and over the malar bones (sometimes very acute, and obviously rheumatic), extreme general uneasiness, contused pains and soreness of the limbs and trunk,—all combined with excessive depression of spirits, and an amount of debility and prostration totally out of proportion with the local ailments (syncope sometimes occurs in the erect posture)—are the symptoms of the established disease in its ordinary and pure form. The physical signs are those of bronchitis, or there are none.

There are exceptional cases (the same poison evidently being at work) where violent headache, flushing of the face, and delirium, with fever, mark its action; others, where the digestive organs alone suffer, and in the upper parts; others marked by diarrhœa or pseudo-dysentery; and yet others where, eretheism or actual catarrhal inflammation of the urinary organs is the main phenomenon. In some epidemics especially, the lung-substance has been very commonly attacked, and pneumonia cut off multitudes of aged and debilitated persons. Pleurisy is also an occasional complication. Both affections are habitually latent, and to be discovered by their physical signs alone.

Terminating, in ordinary cases, by diaphoresis, sometimes by (or rather with) cutaneous eruptions, at the end of from three or four days to a week, influenza leaves after it, invariably, more or less debility, and, in many cases, chronic bronchitic cough. That the symptoms of phthisis have occasionally first become apparent after an attack of influenza, is unquestionable; and the fair inference is that it accelerated the outbreak of the tuberculous disease. Influenza rarely kills those it attacks, unless, aged and debilitated, they have already one foot in the grave. Among this class of the population, the mortality occasioned by an epidemic has sometimes proved extremely serious.

II. The experience of centuries has established, beyond question, the impropriety of depletory measures in the *treatment* of influenza. The Czar of Russia was justified, by the mortality clearly traceable, not only in his own dominions, but in various countries of Europe (England, France, Spain, Italy), to blood-letting, in issuing his Ukase against its employment. I doubt if the occurrence of consolidation-signs even warrants the use of the lancet: such signs are observed chiefly in the aged and exhausted; and I know, from experience, that dry-cupping and carbonate of ammonia will remove these signs in such cases; whereas I have never seen any benefit derived from abstraction of blood, either by leeches or cups. Such consolidation is very positively *passively congestive* in the aged and infirm, and is increased by depressing measures. Should well-marked signs of sthenic pneumonia occur in a young or middle-aged person, leeching or cupping may be advisable;—but even then caution is requisite; a dozen leeches have, within my experience, produced successive fainting fits in a previously healthy, and robust individual. I have never seen a case (perhaps such may be met with in the provinces) where venesection was advisable. Neither should tartar emetic, active purgatives, nor (I think, in spite of the encomiums of Dr. J. Davies) mercury, with a view to its constitutional action, be employed.

The treatment I have found most successful, is as follows:— Keep the patient in bed; open the bowels by some gentle laxative; give some slightly diaphoretic medicine, in combination (if there be rheumatic pains especially) with colchicum and an alkali; procure sleep by extract of lettuce, or of hyoscyamus; and allow diluents freely. After the first three or four days, if bronchitic rhonchi exist, a blister should be applied between the shoulder-blades or to the sternum, and an expectorant mixture prescribed. This mixture may with advantage be made somewhat stimulant;—let the vehicle, for example, be partly ammoniacum mixture, or decoction of senega; lobelia inflata, and paregoric should enter into its composition. Tonics, iron, and quinine may be given daily during convalescence, unless the stomach have been implicated to any amount. If there be much exhaustion from the first, sesquicarbonate of ammonia and strong beef-tea should be given without hesitation.

SYPHILITIC BRONCHITIS.

That the virus of syphilis may affect the bronchi, has been made very evident by the inquiries of Drs. Graves, Stokes, and Munk. It appears that a certain time after infection, febrile action, and bronchial irritation occur in a variable number of cases, as preludes to cutaneous eruption, disappearing wholly or partly when this is established; and, conversely, if a syphilitic eruption suddenly disappear, spontaneously or through treatment, bronchitis may ensue.

Under these circumstances, the diagnosis is easy. But persons, poisoned to the secondary and tertiary degrees by syphilis, may have chronic bronchitis, as a persistent state,—they may cough, have sero-purulent and muco-purulent expectoration, nocturnal perspiration, and hectic fever, while they rapidly lose flesh and strength; and no tubercle shall exist in the lungs. Yet here is assuredly enough to create a strong suspicion of its existence, taken in conjunction with the indubitable tendency of syphilis *plus* mercury to induce the outbreak of phthisis in a person having the requisite constitutional aptitude. How are the cases to be distinguished? By the total want of accordance between the physical signs and the constitutional symptoms: the patient with syphilitic bronchitis has neither consolidation signs, nor, *à fortiori*, those of excavation. But there is a curious source of difficulty, which sometimes starts up in these cases, and renders doubt imperative: the infra-clavicular ribs and clavicle thicken from periosteitis, and produce dulness under percussion, which cannot with positiveness be distinguished from that of tubercle within the lung. Here the observer must wait for events to clear up the diagnosis.*

In managing this variety of bronchitis, the whole secret consists in having proper regard to the diathesis inducing it. Ioduretted inhalations are serviceable.

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* *E. g.* Case of Smedley, U. C. H., Females, vol. i. p. 143 (1846). When admitted in 1846, this woman, in addition to secondary and tertiary syphilis, and cancer of the rectum, had bronchitis and very slight dulness, with harsh respiration under the *right* clavicle; but as the subjacent bones were obviously thickened, I abstained from giving any positive opinion as to the existence of consolidation of the lung. The chest-symptoms totally disappeared under treatment. I have frequently seen the patient since; consolidation-signs, growing at each interval more marked, had become positive at both apices when I last (winter of 1850,) saw her.

NARROWING OR OBLITERATION OF BRONCHI.

Narrowing and obliteration of the bronchi, a common phenomenon in tubes of very small calibre, becomes rarer and rarer directly as their size; still, obliteration, even of the main trunk, has occasionally been witnessed. The obstruction may depend on *intrinsic* causes,—such as thickening of the mucous membrane, accumulated secretions, especially of the plastic kind (under both these circumstances the condition is of inflammatory origin), or an accumulation of tubercle or cancer in their interior; or obstruction may be produced by *extrinsic* pressure,—for instance, that of adjacent emphysema, adjacent tuberculous deposit, plastic contractile exudation, infiltrated cancer or chronic solid pleural accumulations: enlarged bronchial glands, aneurisms and mediastinal tumors have sometimes effected the closure of a main trunk by pressure.

If obstruction of a large bronchus, by its own secretions, occur suddenly, or even with notable celerity, dyspnœa proportional to the size of the tube follows; and as this has chiefly occurred in cases of bronchitis, where the efficient breathing surface had already been seriously diminished, risk of fatal asphyxia is incurred, unless the patient retains strength sufficient to enable him to expectorate the accumulation: the relief experienced after such expectoration is almost assimilable to that following tracheotomy for obstructed larynx.

But if the obstruction be on a small scale, or if a large tube suffer only from slow, gradually increasing obstruction, there will be no positive subjective or objective symptoms to indicate its existence. Such dyspnœa as is really traceable, in part, to obstruction of the kind, appears explicable by other conditions; and neither cough nor pain in the chest exists.

The physical signs may or may not be satisfactory. Obstruction of the main tube produces collapse of the entire lung, as well shown in one of Dr. Carswell's published drawings;[*] obstruction of minor tubes, as insisted on by Dr. Stokes, produces local and limited collapse,—the surface of the lung in the affected parts appearing sunken in below the plane of the surrounding pleura. Now, in both these varieties percussion-dulness, proportional to the superficial extent and depth of the collapse, would constantly exist, were it not that, in the local variety, emphysema tends to spring up on the confines of the collapsed spots, and give rise to its own special resonance. The respiratory murmurs are either actually suppressed or weakened considerably, and harsh; and mingled with sonoro-sibilant rhonchi.

The main interest, in the present state of knowledge, of obstruction of a large bronchus, comes of the light it may throw on the diagnosis of thoracic aneurism and tumor.

The treatment of the affection is altogether that of the disease of which it is a sequence or effect.

PLEURISY.

The physical signs of pleurisy, and to a less degree its symptoms, vary with the anatomical conditions of the disease—themselves referrible to four periods: that of hyperæmia and dryness; of plastic exudation; of sero-purulent effusion, without or with dilatation of the side; and of absorption, without or with retraction of the side.

Physical Signs.—Dry Period.—The motions of expansion and of elevation, from the consensual avoidance of pain, are diminished to the sight, feel, and measure; their rhythm is slightly jerking. The percussion-sound is not perceptibly duller than natural; I have never known its pitch raised. The respiration is weak, but superficial, and jerking in rhythm. Grazing friction-sound may sometimes be caught, especially about the infra-mammary and infra-axillary regions.

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[*] Fasc. Atrophy, pl. iv., fig. 3. The lungs are those of a monkey,—the cause of obstruction, extensively tuberculised bronchial glands. Atrophy from inaction would doubtless ensue after a time.

Plastic Exudation Period.—The state of chest-motion continues as before; rubbing vibration may sometimes be felt with the hand, but is very rarely perceptible to the patient: the vocal fremitus continues natural. The clearness of percussion-sound is somewhat diminished; if it be at all notably impaired, and the sensation of resistance be even slightly increased, the plastic exudation is abundant: deep inspiration will restore the sound to its natural character. The respiration continues of weak, jerking type; and is accompanied with friction-sound of the rubbing or even grating varieties: pleural friction of cardiac rhythm may also by possibility be detected. No influence is exercised on the resonance of the voice: if pseudo-ægophony exist, it depends on some condition extraneous to the pleura.

Period of Effusion.—(Without dilatation of the side.*) The jerking rhythm of the motions visible in the past stages now gradually disappears; and both the expansion and the elevation classes of movement (the latter are less affected than the former) cease to be visible at the lower part of the side: the infra-mammary and infra-axillary regions become more or less bulged. The intercostal spaces, especially in the infra-lateral regions, are less hollow than on the healthy side, and sink in much less with inspiration. Vocal vibration is absolutely abolished, wherever fluid has accumulated to any amount: above the fluid, it continues perceptible: so, too, friction-fremitus, if it existed before, disappears wherever the fluid prevents the collision of the layers of exudation-matter; but may remain in full energy above the level of the effusion, or continue in front, while annulled in the back. Mensuration proves the existence of deficient expansion and retraction; but the difference of respiratory play on the affected and non-affected sides is not so great at this period of pleurisy as in more chronic conditions,—pain still interferes with the general chest-action, and the non-affected lung has not yet acquired the habit of expanding to its full limits. Meanwhile dulness under percussion becomes very manifest, with increased parietal resistance, inferiorly; the limits of the dull and clear sounding parts are distinguished by a tolerably well-defined line,—and the area of the dulness, and its line of demarcation, may be changed by altering the patient's posture, but not by the act of respiration. In rare instances the inferior lobe of the lung loses bulk so considerably by pressure, that a fall may occur in the upper limits of dulness, in consequence of the fluid gravitating downwards to fill the otherwise vacant space; hence the area of superficial dulness may by possibility decrease, while the fluid actually increases. Auscultation shows that the respiratory murmurs, suppressed where the effusion is most abundant,—weak, and deep-seated, where less abundant, acquire, above the level of the effusion, exaggerated force, and more or less of harsh, bronchial, or even blowing quality. Friction-sound may still be sometimes discovered above the confines of the fluid. This is the most favourable period of the disease for the detection of ægophony, as already (p. 134, *et seq.*) fully explained: but the vocal resonance may be strongly or weakly bronchophonic, or may be absolutely null; the cases in which ægophony is actually caught are certainly in the minority,—a fact for which an explanation has already been tendered. The heart's sounds are heard with greater intensity through a given thickness of fluid, and of lung solidified by condensation, than through an equal thickness of healthy parenchyma; hence, in right pleuritic effusion, they are more clearly audible than in the natural state, in the right-axillary region. Besides, the action of the heart produces a sort of succussion or fluctuation-movement in the fluid, which (more especially if the organ be hypertrophous) is very perceptible at the opposite surface of the chest.

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* M. Woillez's division of the effusion-period into two sub-periods, the *laminar* and *gravitating*, though founded so strictly on physical principles, that there is great difficulty in contesting its justness, may be ignored in a practical work, because clinically it cannot be substantiated; at least, I have never succeeded in finding the signs of a thin sheet of fluid equally spread over the entire lung, from apex to base. The time during which the suction of the lung is a force more powerful than gravitation of the fluid must be exceedingly brief.

Period of Effusion with Dilatation and Thoracic Displacements.—The fluid having accumulated sufficiently to fill all available space in the pleural sac, increased in capacity to a great amount by compression of the lung, begins to push the walls of that sac before it in all directions. The parietes, the mediastina, the diaphragm, and the triangular apex of the pleural sac above the clavicle yield before the encroaching liquid,—hence various important modifications in the physical signs. The affected side, either expanded generally, or bulged below to a high degree,—motionless inferiorly, unless a powerful inspiratory effort be made, when a slow, dragging, upward movement takes place, posterior in time to the expansion of the other side,—exhibits a notable change in the state of its intercostal spaces; these are widened, flattened, or even convex outwards, and in the latter state may be, besides, the seat of visible fluctuation. The outline of the affected side is felt to be unnaturally smooth; all vocal vibration has absolutely disappeared; and peripheric (more rarely simple) fluctuation may be detected in the distended intercostal spaces. The semicircular, antero-posterior, and vertical measurements of the side, and the distance between the nipple and middle line, increase; while the respiratory play falls notably,—in fact may be null.*

* The respiratory play may be otherwise singularly perverted. Thus, in a case of left effusion now under observation (H. Morris, U. C. H., January 1, 1851), the seventh and eighth ribs and interspaces below the left nipple, sink in during forced inspiration, both visibly and palpably, while the abdomen rises considerably; yet semicircular mensuration indicates at the same spot a respiratory play on the affected side of three-eighths of an inch (Insp. 17¼; Exp. 16⅞ inches). This is a striking discordance between antero-posterior and general expansion movement (vide p. 33).

The dull percussion-sound of the previous period now extends downwards, upwards as far as, and even by possibility above, the clavicle, and passing the middle-line in front, encroaches at the upper part of the mediastinum on the opposite side of the chest. The displaced heart also carries its own proper dulness into the new position the fluid may have driven it. All sense of elasticity in the parietes has disappeared; and the outline of the dull sound is scarcely to be altered by changing the position of the patient.* Such is the more usual state of the percussion-sounds. But in certain instances the upper part of the chest, especially about the clavicle, first and second ribs, and adjoining part of the sternum, even to the other side of that bone, acquires a tubular, amphoric, or tympanitic resonance, or tympanitic and amphoric combined. I have observed the amphoric variety in cases of latent effusion, where the patient has been walking about, and unconscious of local ailment of any kind. The mechanism of these peculiar qualities of percussion is elsewhere considered.† The respiratory murmurs, totally suppressed, except close to the spine and at the apex of the lung, possess in those regions a harsh, bronchial, or even slightly blowing quality,—a quality explicable by the condensed state of the lung furnishing the murmurs. In some exceptional cases—the theory of the fact has already (p. 93) been considered—diffused blowing respiration is pretty extensively heard over the diseased side. Friction-sound, in rare instances, still continues audible in some limited spot; the rule is, that it totally disappears. The continuance either of ægophony or any other form of vocal resonance is altogether exceptional. The heart's sounds may be well heard through the fluid.

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* Even at this advanced period of effusion, alteration of the kind is, however, possible. If a patient with fluid enough in the pleura, when he lies on the back, to render the sound dull to the opposite side of the middle line, be made to lie on the side of the effusion, in the course of a minute or two the sound becomes clear at the mediastinum. I have found this fact of considerable utility in certain cases in determining whether there was or was not mediastinal tumor present, in addition to abundant pleural effusion.

† While these sheets are passing through the press, I have observed a case throwing additional light on the mechanism of amphoric resonance in pleurisy. In a moribund patient of Dr. Garrod's (U. C. H., Ward 7), to whom I was called accidentally in the absence of that physician, and in whom I found, in addition to the more ordinary signs of copious pleuritic affection, *amphoric* (not tympanitic) resonance at the upper and inner part of the side, extending across the first bone of the sternum, the post-mortem examination, a few hours later, proved the presence of air in the site of the amphoric resonance. Air, at all events at the *upper* part of the chest, may then give an *amphoric* quality to the resonance: but the vicinity of the trachea and large bronchi must have exercised an influence on the quality, converting it from tympanitic into amphoric.

The diaphragm, depressed to a variable amount, carries with it the liver or spleen; with the mediastinum, the heart is carried to the opposite side. The amount of lateral detrusion of the heart is sometimes very remarkable: in cases of left effusion, the organ may pulsate outside the right nipple; and thirty-six hours will sometimes suffice to produce this amount of malposition. The visible and palpable impulse of the organ, and the maximum points of its sounds (especially of the first), prove its change of place. Does the displacement exercise any influence on the quality of the sounds? Most unquestionably it does not, in the great majority of cases; my experience on the point perfectly accords with that of Dr. Stokes, who feels convinced that, even when at its height, the displacement "does not cause any alteration in the natural sounds of the organ." But the rule, I am equally persuaded, is not without its exceptions. Larrey has related a case in which extreme feebleness of the pulse in the large arteries co-existed with it; there can be no certainty, however, that there was any mutual dependence between the two circumstances. Hope describes the following peculiarity in a case of right lateral detrusion from effusion in the left pleura: "The aorta was felt to pulsate between the second and third right ribs, an inch from the sternum; and here a murmur was heard with the first sound, which has *ceased* since the heart has been restored to its natural situation by the absorption of the fluid. Is it, therefore, possible that a twist given to the aorta, or pressure of the vessel against the ribs, may be the cause of a murmur under such circumstances?" In a most interesting case recently observed,* for many successive days during the height of left pleural effusion, both sounds of the heart (pushed to the right of the sternum) were more or less masked by blowing murmurs; these murmurs, when the heart was restored, or very nearly restored, to its natural position, almost completely disappeared,—a fact the more remarkable, as, from the slowness of convalescence, plenty of time had elapsed for the formation of the systolic basic murmur of spanæmia. The diastolic murmur disappeared the more perfectly of the two, and must positively have depended (probably through torsion of the aorta) on the displacement of the organ. I have never yet, no matter how profuse the effusion, nor how complete the evidences of *eccentric* or dilating pressure, seen signs of *concentric* pressure in these cases: I should regard the detection of the latter class of signs, in a case of pleuritic effusion, as proof positive that there was some additional disease (tumor, aneurism, &c.) within the chest.

Period of Absorption.—The absorption of pleuritic effusion is effected, with results of two kinds, on the form and condition of the chest. The walls may be simply restored to their natural position, or they may sink inwards,—inside, as it were, their natural site. Hence, clinically, we have a period of absorption *without* and *with* (the former infinitely the more favourable of the two) *retraction of the side.*

Without Retraction.—The visible enlargement and bulging gradually disappear, and with them (but very slowly) the obstructed state of the chest-motions; the natural intercostal hollows (deepened, perhaps, even by emaciation) are again felt; friction-fremitus and vocal vibration return,—the former often with greater intensity than at the outset of the disease. The various measurements fall to their natural standards. The percussion-sound gradually recovers its natural clearness, first at the upper, then at the lower, regions; in the latter, indeed, it may long (for weeks) remain dull from imperfect expansion of the lung, and accumulation of false membrane in the pleural sac. The respiratory murmurs, gradually restored, remain for a variable time weak and harsh or bronchial; friction-sound re-appears (or *pleural pseudo-rhonchus*) for a variable period; ægophony is sometimes caught passingly for a day or so, but the vocal resonance quickly becomes bronchophonic, or may be null. The heart, with the mediastinum, returns to its natural position, sometimes with singular quickness. I once saw a heart beat in its natural position, which, *seventy* hours only before, I had felt pulsating under the right nipple; absorption so rapid, singularly rare under all circumstances, was here the more remarkable, as the patient was tuberculous, and had had several attacks of profuse hæmoptysis.

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* Henry Morris, U. C. H., January, 1851.

Without Retraction.—General retraction of the affected side is very much less common than partial depression,—the latter occurring probably about twelve or fifteen times as frequently as the former: general retraction is a process requiring much time for its accomplishment. From the inquiries of M. Woillez, partial depression would appear to be more frequent in front on the right side, behind on the left side. The shoulder, the ribs, and the nipple, fall (in some peculiar cases the shoulder rises, however); the scapula becomes tilted outwards at its inferior angle; the dorsal spine curves laterally, the convexity looking (at least in the majority of cases) towards the sound side; and the ribs undergo distortion, their external planes becoming more or less inferior. This altered condition of the ribs lessens the width of the intercostal spaces; and in cases of very chronic course hypertrophous enlargement of the ribs still farther decreases that width.* The chest-surface laterally is unnaturally irregular and uneven; in front it may be flat, smooth, and even slightly concave, the distinction between ribs and interspaces being completely lost to the eye. Rubbing vibration is rarely to be felt. All the chest measurements (those of the side, generally, as well as the partial class) undergo diminution, with the exception of that between the clavicle and nipple, which increases.* The measured chest-play, on the diseased side, may be absolutely, or next to absolutely, null, while that of the other side exceeds the individual standard of health (p. 34). The percussion-sound, dull, with marked resistance, inferiorly, acquires the wooden quality at the mid-height of the thorax, and is often tubular at the apex, in front and behind. The enlargement of the ribs, just referred to, contributes to increase the dulness of the sound, especially laterally. The signs obtained by auscultation, though less striking than these, are sufficiently important; the respiratory murmurs more or less completely suppressed at the extreme base of the side, are superiorly weak, bronchial or diffused blowing, — months may elapse, after retraction has commenced, before respiration is restored to any extent; friction-sound, of creaking or grating type, may or may not be audible; the voice resounds with morbid intensity, especially at the central parts of the side. The vault of the diaphragm and subjacent viscera are sometimes drawn above their natural level, — an elevation not faithfully indicated by changed position of Harrison's sulcus. The position of the heart varies in at least five different ways:—(a.) The organ having slowly or rapidly, gradually or suddenly, retraced its steps, recovers either its natural situation, or the immediate vicinity of this: here is the most common case. (b.) It remains in the abnormal position into which it was forced by the effusion, in consequence of the establishment of adhesions. (c.) The tractive force may be so powerful at the period of absorption as to *pull* the organ out of its place in the converse direction to that into which it had previously been *pushed* (an occurrence best observable when the right pleural sac has been affected.* (*d.*) It may happen, according to an observation made by Dr. Stokes, that the heart hangs more loosely than natural in the chest, and so falls somewhat to the right or left, as the patient lies on this or that side. (*e.*) Or it may happen, in cases of left effusion, that the heart, originally pushed enormously to the right, subsequently passes to the left, even beyond its natural site,—and still later, regains more precisely its normal position by repassing a very little to the right.†

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* U. C. Mus., No. 4001. The ribs in this specimen are not only enlarged by true interstitial hypertrophy of (especially) the cancellated structure, but by adventitious bone-formation between the periosteum and original ribs.

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* This comes of lowering of the nipple,—a more valuable sign on the right than the left side.

The physical signs, now described, are those of the disease from the moment of attack to its final term,—in other words, of its acute and chronic forms. The symptoms of each form must be separately considered.

Acute form.—Pleurisy almost always commences with rigors—proportional in severity to the intensity of the coming inflammation, but very rarely as marked as those of pneumonia. The rigors may be either actually the first symptom, and anticipate pain by some hours or even days, or be preceded for a few hours by the characteristic "stitch in the side." This peculiar pain, dragging and shooting in character, increased by movement, pressure, and percussion, varying in severity from a mere annoyance to a feeling of agony, commonly seated below the nipple, near the antero-lateral attachments of the diaphragm, and under the scapula, rarely extending over the entire side, still more rarely limited to the confines of the abdomen, or the abdomen, is persistent, temporary or intermittent, and sometimes totally disappears (more commonly remits) with the occurrence of effusion. Increased frequency of breathing is even a more constant symptom than local pain,—it may be unnoticed by the patient, or attended with very perceptible dyspnœa. The number of respirations very rarely exceeds the third of the number of the heart's pulsations;—indeed, I do not remember ever to have observed, in an uncomplicated case, a ratio of the latter to the former lower than 3:1. It is most important, however, to distinguish such dyspnœa from the mere spontaneous hastening of breathing, which sometimes comes of the petulance of pain. The breathing-motion, as we have seen, is restrained in amount on the affected side; its increased frequency acts as a sort of compensation.* The dyspnœa is generally more marked at the outset, than after effusion has occurred. Cough exists in the great majority of cases, dry (unless there be co-existent bronchitis), short, small, suppressed, and frequent. Tenderness of the intercostal spaces, under pressure, is sometimes highly marked;—that it depends on the state of the pleura itself, is shown by the fact that no pain may be excited until it is inferrible, from the force employed, that the serous lamina is directly pressed upon. During the dry and plastic stages, the patient commonly lies on the sound side or on the back; I have seen exceptional individuals lie by choice on the diseased side, to control motion and stifle pain, as they assured me: generally speaking, this posture increases pain. After effusion has occurred, the patient lies on the back, on the affected side, or diagonally between both, with the head somewhat raised.

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* *E. g.* Case of Griffiths, U. C. H., Males, vol. ii., p. 174.

† Case of Luckett, U. C. H., Clin. Lect., loc. cit., p. 390.

The general symptoms are those of a febrile inflammation. The pulse is frequent, sometimes hard and concentrated; the skin, hot, but not acridly or burningly so, at the outset, becomes moist at the effusion-period. There is but slight prostration of strength; and the cerebral functions are very rarely affected. The urine, of febrile character, (high specific gravity, deep colour, and strong odour,) may be temporarily albuminous, as in other acute affections, and has been found, in rare instances, to contain fibrine. The blood is hyperinotic,—the fibrine ranging from 3·5 to 7 per 1000: as a rule, it ranges lower than in pneumonia. The buffy coat is absent from blood drawn in about one fourth of cases.

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* This restraint of motion on the pleuritic side is a remarkable instance of consensual action, or of motion regulated involuntarily by sensation. It cannot be imitated by the will, and is therefore involuntary; it depends on sensation, and is therefore not reflex.

The terminations of acute pleurisy are by recovery (resolution or absorption); by lapse into the chronic state; and, under certain circumstances, by death. Death is so rare a result of the disease, when attacking individuals free from organic affections, that I have neither myself (and I have carefully attended to the point, since my attention was first drawn to it, years ago, by M. Louis) lost a patient from pure primary idiopathic pleurisy, with or without effusion, nor known of an occurrence of the kind in the practice of others. And, although, where chronic disease either of the lungs or of other organs pre-existed, death is a more common result, it is still an unusual one. Pleurisy is rarely the immediate cause of the fatal event in phthisis; it is only so by rare accident in chronic bronchitis; and, although *both* pleuræ suffer in blood diseases attacking the serous membranes (as, for instance, in uræmia and pyohæmia,) pleuritis is even here not only an uncommon apparent cause of death, but when apparently destructive to life is generally conjoined with pneumonia. I have, proportionately to the rarity of these diseases, found the secondary pleurisy of carcinoma of the thorax and its contents * the most fatal variety.

Chronic pleurisy presents itself clinically in three conditions or forms:—(1) With retraction of the side, and the various accompanying physical imperfections already described: (2) With permanent dilatation of the side by sero-albuminous or purulent fluid (*empyema*): (3) With permanent fistulous opening in the pleura, and discharge of pus from the sac.

In the first case of retracted side, the rule is, that the general health is below par,—the individual is thin, incapable of much physical or mental effort, and prone to slight passing attacks of inflammation in the affected side. The breath is rather short, and dyspnœa easily evoked; there is frequent pain in the side.

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* Dewing, vol. v. p. 19. Unwin, U. C. H., Males, vol. iii., p. 289.

But in exceptional cases, hypertrophy of the fellow lung occurs on an extensive scale, and the patient does not, *quoad* facility of breathing (breathing power is another thing), differ notably from healthy persons.

In the second case (*empyema*) of distension with sero-flocculent or purulent fluid,—conditions of the fluid which cannot be distinguished with certainty during life,*—symptoms of more prominent character exist. The patient either lies on the back, or diagonally on the diseased side, with the head slightly raised and bent towards that side; he rarely suffers from local pain, unless some intercurrent acute inflammatory action arise. His dyspnœa varies in amount; I have never known it seriously pervert the ratio of the pulse to the respiration. The voice is weak; the cough frequent, either dry or attended with expectoration, muco-purulent or purulent. Not only does empyema not give rise in itself to hæmoptysis, but empyema, established in a case of phthisis, appears to a certain extent prophylactic against the hæmoptysis, which is almost an appanage of the latter disease.† Expectoration habitually comes of some coexisting affection of the lung or bronchi,—it is steady in amount, and rather abundant. But sometimes sudden profuse pouring forth of sero-pus by the bronchi takes place, through perforation of the pulmonary pleura,—or a similar discharge, to the amount of two pints or more, may rapidly take place without any such perforation occurring (at least so far as trust can be put in physical signs, and commonly they are absolutely conclusive on the point), but simply through a metastatic flux from the bronchi. Œdema of the affected side of the chest is common; it may extend to the corresponding limbs, and half of the abdomen. The face is puffy, and semi-transparent, without malar flush; the lips tumid and livid. The external thoracic veins are not enlarged, the eyes are not prominent, there is no dysphagia, and no stridulous character in the voice or cough: in a word, there are no signs of concentric pressure. The opposite lung becomes hypertrophous and emphysematous; sometimes its bronchi (rarely its parenchyma) inflame. The pulse is habitually frequent, quick and small; the patient easily falls into a state of syncope; the skin is hot and dry; the febrile action of hectic type (and this whether the contents of the pleura be pure pus or not); anorexia and insomnia combine with other causes to produce serious emaciation.

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* Bulging of the intercostal spaces was once supposed to occur only where the contents of the pleura were purulent;—a most unquestionable error, as is now commonly understood.

† For the facts on which this proposition is founded, *vide* Author's Report on Consumption, loc. cit.

The terminations of empyema are by death (through slow asthenia); by recovery, through absorption, or through evacuation of the fluid by the bronchi or parietes,—the opening undergoing closure after it has served its purpose; or by lapse into the fistulous form of the chronic disease.

There may, under the latter circumstances, be one or more fistulæ, and the discharge may be trifling in amount, or sufficiently abundant to account in itself for emaciation. In these cases, retraction of the affected side is carried to the extremest amount possible, and hypertrophy of the opposite lung * reaches its maximum. The symptoms in this variety of the disease are habitually similar to those of the form last described. In rare instances, not only is life prolonged without obvious suffering, but the individual is able, with care, to follow his ordinary pursuits; death in the great majority of cases is the slow result.

The proportion of cases of acute pleurisy, lapsing into the

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* The lung in this state, instead of receding, when the chest is opened after death, may actually protrude through the opening. I have known the difference in the semi-circular measurements of the sides, equal four inches in a case of the kind. Increase of bulk of the sound lung takes place, with tolerable speed, too. I have established (case of T. Wicks, U. C. H., Males, vol. i., p. 59, 1847,) by measurements made at an interval of eight months, an increase (emaciation having all the while advanced) equalling very nearly two inches in the semi-circumference of the side.

varieties of the chronic disease, is unknown: pre-existing organic disease of the lung predisposes to this course.

II.—The *diagnosis* of pleurisy is in the majority of cases sufficiently simple; but at all its periods there is a certain chance of confounding it with other diseases.

In the *dry* period, pleurodynia (with bronchitis accidentally co-existing) and intercostal neuralgia are the two affections most easily mistaken for pleuritic seizure. The rules for distinguishing them have already (p. 255) been given.

In the plastic stage, pleurisy may, under ordinary circumstances, be distinguished from plastic pericarditis by the respiratory rhythm of its friction-sounds. In those comparatively rare cases, where the heart's action produces friction of its own rhythm within the inflamed pleura, while the pericardium is perfectly free from disease, attention to the rules already laid down (p. 235), will generally remove all difficulty in the diagnosis. The course of events will soon settle the question, should doubt remain in spite of the aid of those rules: if the friction of cardiac rhythm be pericardial, the signs of pericardial effusion will, with almost absolute certainty, quickly follow.

Friction in the peritonæum is, especially at the upper part of the abdomen, with difficulty distinguished, as its rhythm is respiratory, from similar sounds in the lower part of the pleura. Indeed, I know of no positively distinctive character, except the locality of the sound; if this be obviously beyond the confines of the chest, and if there be no such friction in the pleura as to account for rubbing-sound being heard under the abdominal walls, as an effect of mere conduction, its origin in the peritonæum must be admitted.* Both kinds of sound may or may not be accompanied with friction-fremitus.

Pleuritic effusion, when acute, may be confounded with pneumonic solidification. But in cases of effusion, vocal fremitus disappears; in hepatisation, it is maintained at, or raised above, the average of health.* In effusion, there is no crepitant rhonchus; nor is there ever true tubular, sniffling, metallic respiration: such blowing respiration as occasionally occurs in pleuritic effusion is of the diffused variety, deep-seated, except close to the spine,—and slight in amount as compared with the amount of percussion-dulness present. Vocal resonance is null, or weakly bronchophonic; in pneumonia, it is strongly and snifflingly bronchophonic. In effusion, the heart and intercostal planes are displaced, and dulness extends across the mediastinum. It is true, the same extension of dulness beyond the middle line occurs to a slight amount in some cases of pneumonia; but where this is the fact in hepatisation, change of position does not affect the mediastinal dulness; whereas it does affect this, as already explained, in cases of effusion. The absence or presence of rusty sputa, of acrid heat of skin, and, above all, the state of the pulse-respiration ratio may be appealed to for further aid; an amount of pneumonic solidification, so great as supposed, will produce a ratio of 2 : 1 or 1·5 : 1. I have never known such perversion result from mere effusion, and the ratio *may*, with copious accumulation, fall within the natural limits. Those rare cases of pneumonia, where vocal resonance and respiration-sound are completely deficient, will be considered hereafter.

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* Case of T. Barker, U. C. H., Males, vol. iv., p. 55.

A much enlarged liver, extending upwards, is distinguished from effusion by the non-protrusion of the lower intercostal spaces (they are, however, more prominent than those of the sound side), by the clear percussion-sound superiorly, and by the tolerably full amount of respiration audible at the posterior base of the chest. The interlobular fissure of a merely enlarged liver maintains its natural relationship to the middle line of the body; that of a liver pushed down by pleural effusion (as Dr. Stokes has shown) lies at an unnatural angle with this. If an enlarged liver displace the heart, it does so in an upward, not, as pleuritic fluid does, in a sideward direction. Deep inspiration increases the area of clear percussion-sound inferiorly, and also that of vocal fremitus, in cases of hepatic enlargement;—it exercises no such influence when dulness depends on effusion.

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* This guide is of comparatively little service, when the left side is affected; the vocal fremitus is naturally so weak on that side: luckily for purposes of diagnosis, mal-position of the heart is most readily effected by effusion into the left pleura. The fremitus-sign is almost valueless in persons with very feeble voices, whether naturally so or from disease. Besides, in cases of *very extensive* and *very dense* hepatisation, the fremitus may be impaired in strength, though not to the same extent as with an equal amount of dulness from effusion (*vide* p. 23). Vocal fremitus may be caught *along the spine* in some cases of abundant effusion.

The spleen, when sufficiently enlarged to increase the width of the left base of the thorax, raises the heart; does not, like effusion, push it aside; extends far into the lumbar region, and forwards to, or beyond, the middle line of the abdomen, with a firm, smooth, or nodulated, surface; affects but very slightly the amount of respiration at the posterior base of the chest; and causes no protrusion of the intercostal spaces. Leucohæmia, if present, will confirm the inference otherwise deduced as to the existence of a certain kind of splenic enlargement; but its absence will not exclude the idea of other enlargements.

The distinctive marks of hydro-thorax, intra-thoracic tumor, and cancerous infiltration of the lung, will be described with those diseases.

Tubercle. in its ordinary seat, at the upper regions, cannot be confounded with effusion, which accumulates below. If the entire lung be solid from tuberculous disease, softening and excavation signs exist superiorly, and the progress of the signs is from above downwards; in effusion, they advance from below upwards. Dulness is never as absolute in phthisis as in effusion: some amount of resilience of the parietes remains too. Mensuration proves the existence of diminished bulk in phthisis, of increased width in effusion. The heart, if displaced in tuberculous disease of the left lung, is carried upwards; in effusion of the left pleura, to the right side; in tuberculous disease of the right lung, the heart may be drawn to the same side; in right effusion, the heart is pushed to the left. Tuberculous disease of an *entire* lung does not exist without implication of its fellow; any amount of effusion may exist in one pleura, the other remaining unaffected. In effusion, the respiration is null or weak, distant and diffused blowing; in phthisis, superficial, of various qualities, and attended with rhonchi.

The signs of the absorption period of pleurisy, in active progress, cannot be confounded with those of any other disease. Pleural pseudo-rhonchus and pseudo-crepitation in the lung, from unfolding of its shrunken substance, can with very little care be distinguished from true pneumonic crepitation, either primary or redux.

Pleurisy, at the period of absorption with retraction, can only be mistaken for affections attended, like itself, with diminished bulk of the affected side,—viz., tubercle, chronic pneumonia, so-called "cirrhosis" of the lung, and infiltrated cancer. Where tubercle diminishes the measurements of the side, it does so mainly superiorly, pleurisy mainly inferiorly: the respiration-motion is greater in tubercle than in chronic pleurisy with retracted side. In tubercle, both lungs are diseased; in chronic pleurisy the organ of the unaffected side may grow extra-healthy, hypertrophous. The signs of softening of the lung are, of course, wanting in the pleural affection. The distinctive signs from the other diseases named, are given with these.

III.—The *treatment* of pleurisy varies materially in its *acute* and *chronic* stages.

(a) In the *acute* disease, especially if the febrile action be of sthenic character, venesection to an amount proportioned to the urgency of the symptoms, and the strength of the individual, should be employed; from a healthy male adult, from twelve to twenty ounces of blood may be taken with propriety. Local depletion by leeches, or better by cupping, should follow this, if the pleuritic pain continue; and in cases of moderate severity may be the only blood-letting employed. I have not observed any more favourable results in cases where blood-letting was pushed to extremes, than when used in moderation; the risk of anæmia and protracted convalescence should not lightly be incurred; and, on the other hand, the time for absolutely arresting the disease in its course has generally passed when the patient comes under observation.* Next in order to blood-letting (free evacuation of the bowels having been effected) comes mercurialisation—to a slight amount—of the system: the more rapidly this influence is produced, the better; and hence, for the first six hours, small doses of calomel and opium (a grain and a half of the former to a sixth of a grain of the latter, or more, if the pain continue acute) should be given every half hour, while mercurial ointment is rubbed, every fourth hour, into the affected side, especially near the axilla. A patient thus treated should be watched from hour to hour almost, and the moment mercurial action exhibits itself, the mineral should be stopped: ptyalism is *not* the object to be attained. After some twelve or twenty-four hours, small quantities of tartar emetic, or of James's powder, may usefully be combined with the mercurial, if (which is rare, on the plan described) mercury has been required so long. Small doses of opium and ipecacuanha at night allay cough and irritation.

As soon as depletion has been pushed as far as is intended, and the febrile action has more or less completely yielded, a large blister may be applied to the affected side, but not precisely to the mainly affected part; its influence over yet lingering pain is sometimes most remarkable, and it probably disgorges the subpleural vessels, by causing effusion from them, and by loading those of the skin. The application of a blister *over the seat of friction-sound* sometimes, it is true, removes this within a few hours, but too rapidly to justify the idea of absorption having occurred: probably a small quantity of fluid, thrown out beside and within the layers of plastic lymph, renders their collision noiseless.*

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* A boy (Henry Falkness) was admitted in the evening of January 10, 1851, (U. C. H.) with acute pain in the side, and dyspnœa. My assistant, Mr. G. Webster, finding marked friction-sound on examination, ordered twenty leeches to the spot. The next day I could not detect a vestige of friction; effusion-signs neither occurred then, nor at any time, (the boy leaving, perfectly well, ten days after admission,—he was detained thus long for purposes of observation). About the fourth day a shade of grazing friction-sound was caught in the old spot. Were exudation and effusion absolutely prevented here by treatment; or was the case one naturally inclined to stop short at the *dry* stage? The friction-sound appears to have been almost too intense for the latter hypothesis.

If, in spite of these measures, fluid continue to accumulate—or if that already thrown out remains stationary—a succession of flying blisters (not kept on each for more than six hours), and friction with ioduretted liniments, should be employed externally, while diuretics are steadily administered internally; nitrate, acetate, and bitartrate of potass, squill, nitric æther, juniper, infusion of digitalis (particularly if the pulse remain frequent) are the agents of this class commonly used. I believe the compound tincture of iodine, in scruple doses, freely diluted, to be a valuable medicine at this juncture. The action of any of these medicines is facilitated by the exhibition of blue pill, digitalis, and squill, every night or every second night.

The diet should be low, and if there be effusion to any amount, much drink should be abstained from: enough will be taken with the saline diuretics employed.

Paracentesis appears to me inadmissible while the disease is acute; if independent of pulmonary tubercle, the chances are infinitely strong that the effusion will disappear by medical treatment; if there be tubercles in the lungs, the mechanical removal of the fluid will not cure the disease. In certain rare cases, fluid accumulates with such rapidity as to threaten life by asphyxia: these cases are certainly fair subjects for exceptional management, and the operation has been performed in such with good results.

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* The late Dr. Macartney, of Dublin, taught that flogging sometimes produced pleurisy in schoolboys (an opinion they would doubtless wish were generally made known to schoolmasters): if the fact be so, it would illustrate the local effect of blisters referred to in the text. Upon the more or less close proximity of a blister to an inflamed surface, will probably depend its exercising an *antagonistic* or *sympathetic* effect.

(b) The *chronic* disease presents itself practically under three main conditions: firstly, the thorax is retracted, the lung incapable, the side painful, the general health impaired; secondly, the side is retracted, its wall fistulous, and a more or less profuse discharge of pus is habitual; thirdly, the side is permanently distended with fluid (*empyema*).

In the first case, art can do no more than palliate symptoms as they arise, and support the strength on general principles. Fortunately in some such cases all local inconvenience ceases after a time, and moderately good, if not robust, health is obtained. The wasting suppuration in the second case points to the necessity of tonic treatment: quinine, iron, and cod-liver oil, are essential to the support of the individual. Efforts may be made, by injecting the sac with slightly stimulant fluids, to alter the character of its secreting surface, and perhaps even induce its sides to cohere: ioduretted fluids are the best suited for the purpose.

In the management of the third condition, that of *empyema*, three indications present themselves: (1) the control of febrile action; (2) the promotion of absorption; (3) the support of the general health.

(1). If antiphlogistic measures have not been put in force with sufficient energy during the acute stage; if febrile action, accompanied or not with local pain, exist; and if the constitutional powers appear not to be severely depressed,—general bleeding, to a limited extent, may be cautiously had recourse to. But it is easy to do mischief by venesection; even under the circumstances supposed, an amount of depression will occasionally follow moderate loss of blood from a vein, which may not easily be recovered from.

The application of some six or eight leeches once or twice a week to the side, for a week or two, is not open to the same objection; and even where local pain and febrile action are totally absent, much benefit may be obtained by the practice, provided the effusion be not of old standing. Or some three or four ounces of blood may be drawn on each occasion by cupping.

Is mercury advisable at this period of the disease? Certainly not, if it have already been used in the earlier stages; certainly not, if there be much wasting of the system, or if there be reason to fear that the fluid in the pleura is purulent. Under other circumstances, a cautious trial of mercury by the mouth and by inunction ought to be made.

(2). Should the disease not yield to these measures, the promotion of absorption is to be further attempted by the use of medicines acting on the secretions; or the use of such medicines may be commenced, while the foregoing plan is pursued.

Diuretics should be exhibited in various forms: the salts of potass, especially the bitartrate and acetate (the former in half-drachm doses, or upwards), and infusion of digitalis, may be commenced with. Digitalis both acts more efficaciously as a diuretic, when administered in infusion, and appears less prone to produce those poisonous effects which occasionally result from its accumulative action. The compound tincture of iodine, largely diluted, is now, as earlier, a very valuable agent of this class. The sustained use of *purgatives* is perhaps deserving of trial, if diuretics fail; but their action, being of a more depressing kind, requires to be carefully watched. They should be very cautiously exhibited when the lungs are tuberculous, either because the intestinal tunics already contain tubercle, which may thereby be more speedily brought into the stage of softening, and induce ulcerative destruction, with its consequences, of the mucous tunic; or because the irritation of the bowel in a person already phthisical accelerates the deposition of tubercle there. I have seen more than one case in which uncontrollable diarrhœa, brought into existence under the former circumstances, evidently hastened the patient's dissolution. On the other hand, dyspnœa, which has resisted various other measures for its relief, may occasionally be removed or materially relieved, and this almost instantaneously, by an active hydragogue. I have observed this from the exhibition of elaterium, even where no manifest change in the thoracic physical signs followed. *Diaphoretics* may be employed, particularly if there be occasional febrile action; but in neither these medicines nor in *expectorants*, as promotive of absorption, can any confidence be justly placed. *Counter-irritation* of the affected side has been employed in every variety, from the mildest to the most severe. The only contra-indication to the use of agents of this class, is the presence of fever. No matter what precautions be taken, their very common tendency is to increase this, where it exists, and hence to promote a condition extremely unfavourable to the advancement of absorption. "First remove feverish action, and then counter-irritate," is a maxim of primary importance. The application of *blisters* exercises a very manifest influence on effusions, when had recourse to with the caution just stated. They should be repeated frequently, made of large size, applied in different situations, not kept on longer than is just sufficient to produce vesication, and every means used to secure rapid healing of the blistered surface.* Employed in this way for some time—say a fortnight—blisters are often most signally beneficial; and I am the more anxious to state emphatically their utility in these cases, because the incredulity as to their efficacy in *acute* parenchymatous and membranous inflammations, now arising among the profession, might possibly be extended to their action in *chronic* maladies of the kind at present under consideration. Should any objection to the use of cantharides exist in particular cases, the tartar-emetic ointment, or a Burgundy-pitch plaster, sprinkled with tartarised antimony, may be substituted; but these are much less advantageous applications. Issues, setons, moxas, and the actual cautery, have all had their advocates and employers in the very advanced stages of the disease.

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* Of all such means, dressing with cotton wool is the best.

The moxa is probably the most advisable of these, both positively and negatively, and may be had recourse to when the use of blisters has failed to affect the disease. The moxa will be comparatively most beneficial when the effusion is of very long standing, and attended probably with anatomical change in the bones and ligaments.

(3). When the disease has become decidedly chronic, the general health is to be sustained by a succulent non-stimulant diet. Strong broths, meats of easy digestion, jellies, &c., may be taken with a degree of freedom proportional to their observed effects; and to this increasingly liberal diet may be added the use of cod-liver oil and gentle tonics, such as tincture of humulus combined with hyoscyamus. If this be well borne, the various preparations of iron, especially the syrup of the iodide, should be employed. There is decidedly a stage of the malady, or, perhaps, rather a condition of the organism generally, in which, although some feverish action be present, the patient will be benefited by tonics.

Change of air sometimes exercises a very manifest influence on the condition of patients with empyema,—primarily, it would appear, on the general health, and, through this, on the effusion. The mere fact of change almost seems to suffice; for there may be no positive superiority in the new atmosphere, in which the improvement occurs, to that for which it has been exchanged.

The modes of treatment now described, sometimes fail altogether. The effusion either remains stationary, or increases, and evidently must destroy the patient in the end. Under these circumstances, a resource is still left in the operation of *paracentesis*, —a procedure which, no matter how divided opinion may be respecting its general feasibility, has assuredly been sufficiently often either completely successful, or productive of marked improvement, to justify its being numbered among the valuable gifts of surgery. This is a vague estimate of its utility, it may be alleged; but, unfortunately, such vagueness of expression is unavoidable. In truth, there are no existing data from which precise inferences may be drawn as to the success of this measure, whether considered generally, or in reference to its performance in particular states of the constitution. And this, because observers have contented themselves with merely ascertaining the existence of effusion into the pleura of subjects operated on, without inquiring into the condition of the other organs, and, above all, substantiating the presence or absence of pulmonary tuberculisation. How can a correct general inference be obtained, when patients are clubbed together, who have simple chronic pleurisy, or this combined with carious destruction of the ribs; who have or have not serious organic disease in other regions of the body; whose lungs are sound, or the seat of active tuberculous disorganisation? During the discussion on the subject of empyema, which took place some years since at the Parisian Academy of Medicine, M. Louis well pointed out the importance of taking the general condition of the patient into consideration, when we attempt to estimate the chances of recovery from pleuritic effusion, either with or without paracentesis. And if we admit Laennec to have been justified in advocating the performance of the operation in the case of tuberculous subjects, because even in them it may be the means of somewhat prolonging life, we must aver that the plea on which *he* based this advocacy,—that a natural cure of tuberculous cavities does sometimes arise,—was of rather a visionary character. I strongly doubt that such cure has ever arisen in persons having superadded empyema.

The quality of the effusion appears to influence the prognosis of the operation. Where this has been purulent from the first, success has more rarely ensued than when sero-albuminous. But cases have terminated favourably, wherein the fluid was, if descriptions of physical characters may be trusted to, really purulent. In respect of quantity, the less abundant the effusion, it is said, the stronger the chances of success. Recovery has, nevertheless, followed when the quantity of fluid amounted to several quarts.

Empyema running a chronic course from the outset, is generally indicative of tuberculous disease of the lungs, and is so far an unfavourable species for operation. Yet, inasmuch as the exudation-matter in such cases is very frequently friable and imperfectly plastic, and the lung therefore less firmly compressed and bound down, than when the solid exudation possesses the opposite qualities, one condition conducive to successful issue—facility of expansion on the part of the lung—is secured. But this element of success, it is almost needless to add, is more than counterbalanced by the constitutional state of the patient.

A notion of the probable result of the operation may be had from the condition of the functions in general; if the conservative functions, digestion and nutrition especially, be only moderately impaired, the chances of the patient's recovery are much stronger than under the contrary circumstances. The operation has been much more successful in young persons than in those of mature years.

The determination of the period of the disease most favourable for operating, is a point of very serious importance. Experience proves, as might have been anticipated, that when performed at an advanced period, paracentesis is rarely successful: the local changes which have then had time to arise in the pleura, pernicious as these are, are even less subversive of success than the deep constitutional distress entailed by the disease. And yet, to this period, puncture of the chest is often, I might almost say commonly, postponed; it can hardly be matter of surprise, that in some such cases it has appeared to do little more than hasten death. There can be no question that the fitting time for operation has come, when a tendency, insuperable by medical means, exists either to increase or to non-absorption of the fluid. The practical difficulty is to determine the precise period at which such tendency may be considered to be developed. On the one hand, perseverance in a given mode of treatment has sometimes proved successful, when everything seemed to foretell its probable inefficacy; and on the other, valuable time may be frittered away, and the period at which paracentesis affords real chance of permanent relief but too easily suffered to pass by from the procrastination caused by a very natural anxiety to give more gentle measures a full trial. A thorough consideration of the whole history of the case, as also of the existing local and constitutional states, is required to enable the practitioner to seize the propitious moment for surgical interference. This difficulty is unfortunately not confined to the operation for empyema.

Let us suppose the operation to be determined on; the next point of inquiry is the manner of performing it. As a measure of precaution, especially if any doubt exist as to the precise nature of the case, a grooved needle may, as suggested by the late Dr. T. Davies, be introduced into the pleura: if the expected fluid appear, the operation may be continued with a trochar or a bistoury. The trochar is the more advisable instrument, if the intention be to evacuate the fluid by degrees,—an intention which we believe Laennec to have been right in commending, when the patient is so debilitated as to justify an apprehension that the complete discharge of the fluid might be followed by dangerous syncope; and when paracentesis is merely performed as a palliative in advanced tuberculous cases. Under ordinary circumstances, the integuments and pleura should be incised with a bistoury.

The first point to be ascertained in selecting the place for the performance of the operation, is the adherence or non-adherence of the lung to the chest, and, in the former case, the precise limits of the adhesions. It is scarcely necessary to say, that the site of these must be carefully eschewed. When the entire side is free from adhesion, the common advice of surgical writers has been, that the opening be made at the most dependent part of the antero-lateral part of the chest. But undiscriminating attention to this advice, given with the sole view of securing free egress for the pleural fluid, has occasionally led to perforation of the diaphragm and abdominal viscera; even the kidney has been extensively wounded by operators whose practice is to adhere too scrupulously to such injunctions. Laennec recommended the space between the fifth and sixth ribs a little in front of the digitations of the serratus magnus muscle, as the fittest site for puncture. The importance of opening the fifth instead of a lower space, has been justly insisted on by Drs. Townsend and Stokes, who draw a fair analogical argument in favour of high puncture from the elevated position in which the discharge commonly occurs, when spontaneous or preceded by the formation of abscess. The opening has sometimes taken place even above the clavicle. If the ribs cannot be clearly distinguished on account of the presence of marked œdema, some difficulty may arise in fixing upon the spot for incision. Steady and continued pressure with the pulps of the fingers will sometimes enable the operator to discover the edges of the bones, in cases where this would on first view have appeared altogether impossible.

The place for operation having been duly determined on, an incision of about two inches in length is to be made through the integuments, and the intercostal muscles carefully divided to the same extent. The cutting instrument should not be carried too close to the borders of the ribs, lest the corresponding branches of the intercostal artery be wounded; this statement applies especially to the lower border of the upper of each pair of ribs, along which the superior, and of the two the much larger, twig runs. The division of the costal pleura itself is by some recommended to be performed with a lancet, but such caution seems useless. When the flow of matter announces the pleura to have been divided, the opening in that membrane should be enlarged sufficiently to give free outlet to the contained fluid.

If the operator have been unfortunate enough to incise the pleura over the site of firm, thick, and strongly adherent pseudomembrane, and in consequence no fluid appear, the line of conduct to be further pursued must vary with the practitioner's confidence in the correctness of his diagnosis in other respects. If he still remain satisfied of the presence of liquid effusion, the attempt to evacuate this may be proceeded with; if he be apprehensive of having altogether erred, the obvious course is to dress the wound, and avert, as effectually as possible, the ill effects of the unnecessary suffering to which the patient has submitted. In the former case, surgeons have attempted to tear asunder the pleural adhesions,—a procedure utterly unjustifiable: the only admissible modes of acting are, to enlarge the existing opening with the bistoury, or to make a new one. The opening may be enlarged, if the free border of the membranous mass interfering with the escape of the fluid be ascertained to be perfectly close to the angle of the existing wound; under other circumstances, a new incision must be made.

Two other methods of opening the chest date so far back as the time of Hippocrates. These are, 1, *perforation of a rib;* and, 2, the *formation of an eschar, either by the actual or potential cautery.*

1. The plan of perforating a rib with a trephine constructed for the purpose, fallen into disuse since Hippocrates, has been recently revived by M. Reybard. The object of this surgeon, in recurring to this antiquated practice, was to ensure a solid support for a canula, which he proposed leaving in the pleura, as a sort of drain for, or, at least, means of removing at will, any new accumulation which might arise. A case thus treated, already operated on in the ordinary way, terminated favourably; and neither caries nor necrosis of the bone appears to have complicated its progress. From this single case, it is, however, impossible to draw any safe inference as to the expediency of the plan.*

2. Modern surgery repudiates the idea of opening the chest by an eschar produced with the actual cautery; and we can imagine no advantage gained by substituting caustic potass for

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* Gaz. Méd. de Paris, Janv., 1841.

the bistoury, while numerous very serious objections to such a procedure manifestly exist. Nevertheless, in cases where an external abscess has formed, and in these alone, the opening in the skin may, if the patient be excessively timorous and impressed with a dread of cutting instruments, be made with a piece of caustic potass.

Some writers lay very great stress upon the importance of preventing the occurrence of pneumothorax; and there can be little question that the entry of air through the wound is a circumstance which it would be well, if possible, to prevent. But the efficacy of any plan devised for its prevention is extremely questionable. Thus the modifications of the common canula, constructed for the purpose of preventing the occurrence of pneumothorax, have invariably been found failures. The proposal to puncture the chest under water is worth trial. It seems, in truth, well to prevent the ingress of air into the pleural cavity, if possible; and this because the existence of pneumothorax must interfere with the expansion of the lung; and the contact of air with the diseased surface is likely to entail increase of inflammation and decomposition of the fluid retained or re-secreted by the pleura. Nevertheless, the prevention of pneumothorax does not by any means appear to be a matter of such importance as considerations of this kind might lead us to expect. In numerous instances, where the physical signs of pneumothorax were distinctly detected after the operation, recovery has ensued.

Laennec appears to me, as already stated, to have correctly pointed out the description of cases in which it may be advisable to evacuate the contents of the chest, by degrees, and upon successive occasions. Under other circumstances, experience shows that no apprehension need be entertained of ill effects following complete evacuation. Speculative reasons may and have been, by various persons, adduced in numbers in favour of each mode of evacuation: the important point to remember is, that *experience* is in favour of as complete evacuation as possible. Curiously enough, many of the advocates of successive evacuations are at the same time energetic in denouncing the ill effects of pneumothorax; how they reconcile to themselves the recommendation of a mode of operating which doubles, trebles, quadruples, or even still further increases the chances of the entry of air, does not very clearly appear.

The proposal to ensure the removal of the entire collection of fluid, by drawing off with a syringe any portion of it which gravitates below the opening, is as old as Scultetus. Mr. Jowett and others have proposed modifications of the syringe employed by that surgeon. There does not appear to be any reasonable objection to the employment of these instruments, if they be dexterously introduced and managed; and they may, in some cases, be decidedly useful. Laennec proposed the use of a cupping-glass and exhausting syringe, with the view of drawing off the remains of the fluid, and facilitating the expansion of the lung.

Is it advisable, under all circumstances, to close the wound after the operation? If the contents of the pleura have been completely evacuated, there can scarcely be any motive for leaving the orifice open. If the evacuation have been only partial, still it is better to close the opening than to leave a canula or tent in it, as the constant renewal of the air in the pleura is decidedly detrimental, and may—unlike entry of air at the first—be prevented by immediate closure of the wound. If, however, any species of canula were devised which would admit of the egress of fluid, and successfully oppose the ingress of air, this objection will cease to exist; and the facility afforded by such an instrument, for the removal of any newly-secreted fluid, will be an important motive for its employment.

The changes induced by the operation are, or may be, of two kinds; *physiological*, and *pathological.* The former, which are local and general, are as follow: the diminution of the contents of the chest, is, of course, attended with decrease in the dimensions of the affected side. In a case observed by Dr. Townsend, the semi-circumference, which reached 16½ inches before the operation, had fallen to 13¾ inches on the ninth day after it. The approximation of the pleural surfaces is rendered manifest, sometimes at a very early period, by the occurrence of friction phenomena; the patient is frequently enabled to lie on the affected side on the day, or even a few hours, after the operation; his dyspnœa is immediately relieved; the state of anxiety under which he laboured disappears; and a sound sleep, the first, possibly, enjoyed for weeks, commonly follows the evacuation.

Of the *pathological* results, as they may be termed, of the operation, pneumothorax is the most inevitable: the question of its influence on the ultimate issue of the case has already been referred to. The continuance of secretion from the pleural surface for a greater or less period, and hence the constant renewal of empyema, is of very common occurrence. In rare instances, the characters of the newly secreted fluid remain those of that originally evacuated; in the great majority, they change, the general tendency of the change being to the purulent character. The alteration from the almost purely serous appearance to the purulent is sometimes accomplished in twenty-four hours. When the fluid has been originally more or less completely formed of pus, but of a laudable kind, its conversion into a purulent matter, of bad quality and fetid smell, is not unusually observed. Under these circumstances, the injection of warm water or some other unirritating fluid becomes advisable. Should there be much appearance of putrescency, a small quantity of very weak solution of chloride of sodium may be added. The notion of giving tone to, and diminishing the irritation of, the pleura, by injecting tonic preparations, seems of very questionable justness; nor has the practice been attended with sufficiently favourable results to warrant its general employment. Ioduretted solutions, or one of nitrate of silver, have more *à priori* reason in their favour.

In the ordinary course of things, when the case is destined to end by the patient's restoration to health, the wound or wounds in the thoracic walls gradually close, and cicatrisation is perfect within a short period; but in some cases, the opening, instead of closing, acquires the characters of a fistula, which it retains for a variable period, and daily gives issue to more or less pus.

When spontaneous perforation of the costal pleura has occurred, and a portion of the contents of the pleural cavity escaped through this into the subcutaneous cellular membrane, forming an abscess there, this should be opened without delay, in order to prevent the occurrence of sinuses and burrowing: so well established is this point, that the operation is under these circumstances termed *empyema of necessity.*

Cases of *double* empyema are, as a general rule, unfit for operation, unless evacuation be rendered necessary for the prevention of asphyxia. Should particular circumstances arise, under which paracentesis might on other grounds become admissible, an interval of time, varying in length with the condition of the patient, should be allowed to elapse between the two operations.

Varieties of Pleurisy.—The varieties of pleurisy are exceedingly numerous. The inflammation may be idiopathic or non-idiopathic,—in the latter case depending on some general disease (as typhoid or puerperal fever); on some diethetic morbid state, as Bright's disease; on some adjacent irritation, as that of pneumonia,* tubercle, carcinoma; on some traumatic cause; on perforation of the lung by tubercle, cancer, abscess, &c. Where necessary, the peculiarities of the inflammation, under these circumstances, will be noticed with the diseases causing them. In the present place, certain varieties of the idiopathic disease will alone be considered.

(*a*) Pleurisy is said to be *latent*, when it runs its course without producing decided subjective symptoms,—where there is neither local pain, cough, dyspnœa, nor febrile action. In cases of this kind, effusion may have reached to the clavicle, and driven the heart greatly out of its place, and yet the patient remain utterly unaware that his chest is the seat of disease. He seeks advice, either from a vague consciousness that he is not in his usual health, or for some ailment totally unconnected with the thorax. The physical signs reveal the true condition of things. The necessary treatment (once the disease is detected) is not modified in any important point (except that blood-letting need not be carried at all so far) by this latency of course; the great difficulty often consists in persuading the patient that there is really anything of a serious character to treat. I have repeatedly known persons with copious effusion of this kind follow their usual, more or less laborious, occupations.

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* A slight amount of plastic exudation in the pleura is so common in pneumonia that pleuro-pneumonia and pneumonia may, in that sense, be used as convertible terms; but pleurisy of clinical importance, and producing effusion, does not occur in more than about one-eighth of cases of pneumonia.

(*b*) The portion of pleura inflamed may be very limited. The disease is then said to be *circumscribed.* When the *phrenic* surface is solely affected, the pain is more severe; orthopnœa is said to be frequent,—even with bending forwards of the trunk; the cough is more paroxysmal; there is hiccup, nausea, vomiting, delirium, excess of costal breathing, jaundice (if the disease be on the right side), and, it is said, occasionally *risus sardonicus.*

Inflammation of the *mediastinal* pleura is often accompanied with serous infiltration of the cellular tissue of the mediastinum itself; *pseudo-rhonchus* will then be heard along the sternum.

Pleuritic fluid accumulation, confined by adhesions between the lobes of a lung (*inter-lobar* pleurisy), may simulate a solid mass in the pleura or in the lung itself, or an aneurism. Dull percussion-sound, local bulging, weak or bronchial respiration, and intensified vocal resonance, may exist in all these cases. The pleuritic accumulation lies in the line of the inter-lobar fissure of the lung; the voice may have an ægophonic twang on its confines; there is no vocal fremitus over it; there is neither impulse nor murmur; and the affection has a past history different from the other diseases named. Similar local collections may form with a boundary of adhesions in any part of the pleural surface, and several such may co-exist, forming sacs perfectly independent of, or communicating with, each other,—whence bilocular or multilocular empyema. The adhesions, forming the walls of these loculi, of course unite portions of lung-substance to the surface, and so give rise to various modifications in the physical signs. The general character of these modifications is, that wherever adhesions exist, blowing respiration is heard; and if a portion of lung, of any size (even though condensed), be agglutinated to the surface, the percussion-sound will be clearer than directly over the fluid; the state of vocal resonance varies. I presume that on the right side the vocal fremitus would be retained over such portions of lung.

(*c*) Empyema, forcing its way through the costal pleura, may form one or more swellings under the skin, which rise and fall (if the parietal communication be free) with the acts of expiration and inspiration. This has long been known. But, some years since, Dr. M'Donnell showed, what had not previously been recognised, that a sub-cutaneous purulent collection of the kind may *pulsate* strongly and expansively, and hence simulate an aneurism. The absence of thrill and of abnormal murmur, the presence of the ordinary signs of empyema, and often the situation of the pulsatory prominence, will clear up the diagnosis. For fuller information, the valuable paper of Dr. M'Donnell may be consulted.*

(*d*) But an empyema may become pulsatile under circumstances more singular than these, and still more likely to confuse the practitioner,—I mean where there is no perforation of the costal pleura, and no accumulation of pus under the skin. I have twice,† in cases presenting all the ordinary physical signs of empyema on the left side (with displacement of the heart to the right), seen the inner part of the infra-clavicular and mammary regions close to the sternum, pulsate visibly, heavingly, and with sufficient force to jog the head at the end of the stethoscope,—the aorta, as proved by *post-mortem* examination, being of natural calibre. In both cases, while the side generally was greatly dilated, gentle local bulging was manifest in the site of the pulsation. The circumstances that determined the diagnosis in favour of pulsating empyema, were the absence of *murmur* at the seat of pulsation; the fact that the two *sounds* heard at this spot were very weak, and gradually increased in intensity, as the stethoscope was carried towards the heart; the absence of thrill below and above the clavicles, and of undue impulse in the latter situation; the perfect equality of the radial pulses; and the total absence of signs of concentric pressure. Taken singly, no one of these characters could be held conclusive, but the entire series formed a most serious body of evidence against the admission of aneurism. In both cases, the pulsation disappeared with the absorption of the fluid, and the return of the heart to its natural position: death arose from independent causes. Pulsation conveyed to empyema, and simulating aneurism by its force and heaving character, seems to be merely an excess of that slight fluctuation movement in the fluid which is far from uncommon in ordinary cases (p. 283.)

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* Dublin Journ. of Med. Science, March, 1844.

† The first of these cases was seen (April 3, 1843,) in consultation with Sir James Clark and Mr. Kingdon.

PNEUMONIA.

§ I.—*Acute Pneumonia*, or diffuse inflammation of the proper substance of the lung, is habitually divided into three stages: those of engorgement, of red hepatization, and of grey (or suppurative) hepatization. Dr. Stokes contends that a yet earlier anatomical stage than engorgement, marked by unnatural dryness of tissue and intense arterial injection, exists; and of the correctness of the opinion I, from actual observation, entertain no doubt.

I. *Physical signs—Stage of arterial injection.*—The respiratory murmurs reach the ear harsher, rougher, and sharper than natural from the affected part, provided this be near the surface; if the affected structure be deep-seated, on the contrary, exaggerated respiration, from the intervening sound, but excited tissue, is heard. I have now seen a fair number of cases in which such exaggerated respiration, coupled with febrile excitement and slight pain in the side, were the earliest indications of a central pneumonia, eventually travelling to the surface. M. Grisolle holds that "in the great majority of cases, if not in all, weakness of respiration, often attended with loss of purity and of softness," marks the outset of the disease. Probably the fact is so, if the part actually inflamed be considered solely; but I have been unable to verify it. The same writer states, that he has, in a large proportion of cases, found *weak* respiration in the neighbourhood of already hepatized lung the precursor of signs of consolidation.

Stage of engorgement.—The motions of expansion and of elevation are, if pleuritic pain be present, somewhat restrained; the vocal fremitus maintains its natural standard; the percussion-sound is more or less dull, according to the amount of engorgement; the respiratory murmurs are weak, suppressed, or masked by rhonchus in the affected parts, exaggerated in those at some distance from them and in the opposite lung; the vocal resonance is somewhat intensified, and slightly sniffling in quality. All this is accompanied with the rhonchus pathognomonic of this stage, the true primary crepitant.

Stage of red hepatization.—Whether pure hepatization be capable of producing general expansion of the affected side, has been a theme of constant dispute. M. Woillez, maintaining the negative, justifies his opinion by a reference to the physical relations of the lung to its containing cavity, corroborated by the results of direct mensuration in *two* cases; in neither of these instances was the least degree of expansion detected. M. Grisolle obtained similar results from circular and antero-posterior admeasurement in *four* cases; nevertheless, he believes, upon the evidence of two others, that the inflamed lung may, quite independently of pleuritic effusion, determine "general or partial dilatation." In one of these, slight bulging of the infra-clavicular region (the disease occupied the upper lobe, and especially its anterior part) was detected on the patient's admission, the third day of the affection. This bulging having gradually increased with the progress of hepatization, M. Grisolle considers himself justified in referring its appearance to the inflammation of the lung; the *post-mortem* examination proved the absence of pleuritic effusion. In the other instance, bulging of the infra- and post-clavicular regions was observed to subside gradually with the resolution of the disease. Now, neither of these cases proves the fact of *general* expansion, as admitted by M. Grisolle; but there does not *appear* to be any plausible objection to them as demonstrating the occurrence of *partial* expansion. In a small minority of cases, I have myself found positive, though slight, increase of width at the base of the affected side. The expansile motion of the chest, and even the abdominal movement (through the diaphragm), are lessened in amount on the affected, somewhat intensified on the sound, side,—and this in simple pneumonia with extensive consolidation, quite independently of the influence of severe pain. But the motion of elevation is not by any means so much obstructed, according to my observation, as that of expansion; a circumstance which will perhaps account for the contradictory opinions held on the subject. In certain cases of pneumonia with a slight amount of plastic exudation on the pleural surfaces, friction phenomena are not to be discovered. To what can this be attributed, but to the diminished expansion of the lung and deficient mobility of the thoracic walls?—while, on the other hand, it would be preposterous to explain the diminished motion by the interference of the painless pleuritic exudation supposed. The respiratory play is impaired, not only by want of *expansile*, but of *contractile*, power on the part of the lung: I have known the latter even more deficient than the former. (*Vide* p. 35.)

As a rule, the vocal vibration is increased in intensity; but in cases of exceedingly extensive, and, as it were, massive hepatization, it *may* actually fall below the natural standard. Fortunately this is rare. A vibratile tremor is sometimes conveyed to the surface through the lung from the heart.

The percussion-sound, habitually diminished in clearness to absolute dulness, with greatly increased parietal resistance, may, under certain circumstances of locality of the inflammation, as shown by Dr. Hudson, acquire a *tubular* or even an *amphoric* quality. This will occur if the solid tissue lie over a distended stomach, or between the large bronchi and the surface. In much rarer cases, the percussion-sound may be purely *tympanitic* over the hepatized lung. I have found this at the anterior base on the *right* side.

The respiration is blowing, diffused or tubular, sniffling, metallic, abrupt, and divided in rhythm; exaggerated in distant parts; no crepitant rhonchus is produced in the actually hepatized part, but may be heard on its confines. Bronchophony, sniffling, muffled and intense, sometimes quasi-œgophonic, bronchial cough, and unnatural intensity of transmission of the heart's sounds, complete the list of discoverable signs. Of all these, the intensified vocal fremitus, the metallic tubular respiration, and the percussion-dulness are the most important.

Sonorous rhonchus was regarded by Laennec as an occasional effect of compression of the bronchi by the indurated pulmonary tissue. However caused, such rhonchus is very uncommon in pneumonia, and is probably, as is generally held, the result of bronchitis. I do not remember to have observed it, to any notable amount, except in the pneumonia of infants, in whom, as is fully ascertained, the co-existence of these two inflammations, or the transition of bronchitis into pneumonia, are much more common than in the grown subject. Were Laennec's opinion respecting the mode of production of dry bronchial rhonchus in pneumonia correct, its occurrence would necessarily be very frequent, instead of being singularly uncommon. Possibly, the co-existence of dilatation of the bronchi may have some indirect influence, as previously alluded to, in the production of sonorous rhonchus in pneumonia; at least, it is certain that the age at which the rhonchus is most common—namely, infancy—is precisely that at which dilatation of the bronchi most commonly attends hepatization,—as shown by Dr. Ogier Ward.*

In certain rare cases of pure hepatization, the physical signs, with the exception of dulness under percussion, are wholly wanting; the vocal fremitus is deficient; there is neither vocal resonance nor respiration heard on the affected side. These peculiar conditions seem probably to depend on such extensive closure of bronchial tubes, as to prevent the possibility of *echo* (*vide* p. 94) occurring within them, while the powers of conduction of the lung are, for some at present inexplicable reason, annulled. Pressure on a main bronchus by an enlarged gland or tumor, if of sufficient size to obstruct the tube completely, will produce this effect on the signs; but such pressure is not a *necessary* condition.

A more singular state of things, still, may exist. Tubular respiration may be intermittent,—sometimes existing, sometimes no respiration being audible. I have traced this in one instance to pressure on the main bronchus of the affected side,—obviously acting at various moments with different amounts of force.†

Stage of Interstitial Suppuration.—The signs in the main are the same as those of red hepatization. If the suppurating points communicate with bronchial tubes,—in other words, if plastic exudation (in and about the vesicles) do not cut off such communication, loose liquid rhonchus, more or less thin and high-pitched, according to the completely purulent quality of the fluid in the lung, will be produced. But there may be complete absence of such signs. I saw a remarkable case of this kind in 1847, with Dr. Storrar. There were neither respiration-sounds nor rhonchi audible in front of the chest, directly over lung in a state of yellow semi-liquid infiltration. Dulness, the most absolute, was the only physical sign in the spot.

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* Med. Gazette, 1838.

† Case of Beckett, U. C. H., July, 1850, Males, vol. vi.

The *resolution* of pneumonia may occur, in some very rare instances, before red hepatization has been effected. In the vast majority of instances, the disease has advanced to the hepatized stage before resolution commences. The signs of this favourable event are, first, a change in the quality of the tubular breathing; it becomes less sharp and metallic, more open,—in a word, of diffused blowing type, which serves as transition to a harsh and weak respiration; the bronchophony rapidly loses its peculiar sniffling quality, but holds on to a slight amount for some time; while redux crepitant or sub-crepitant rhonchus, becomes, and remains, audible for a variable period. The percussion-sound gradually grows clear, and, as a rule, in cases of favourable course, more rapidly recovers its natural character than after the absorption of pleuritic effusion. I have known the tone scarcely different from that of health in a spot where, three days before, there had been the most perfect dulness: but such rapid resolution is, unfortunately, very rare. It has been made a matter of much dispute, whether retraction of the chest-walls may occur in the advanced periods of pneumonia, where no suspicion can exist of the presence of pleuritic effusion. Dr. Stokes teaches the affirmative. Contraction may be observed, he states, in cases where the lung has been long indurated and still continues impervious, and may even co-exist with gradual and ultimately perfect resolution. In all cases where he has observed this contraction, the primary disease has been of the *typhoid* type; in one instance of the kind, the contraction seemed to affect the whole side more than is general in pleurisy; in other cases it was very similar to that of empyema. M. Woillez maintains, on the contrary, that where contraction occurs in pneumonia, there has always been some effusion into the pleura—in fact, pleuro-pneumonia,—and that the process of contraction is the same as in simple pleurisy. M. Grisolle adopts the same views respecting this question as M. Woillez. In nine cases of *simple* pneumonia, antero-posterior and circular admeasurement failed to detect any diminution of size during the progress of convalescence.

Some years since, I observed a case of extensive pneumonia of the left side, in which indisputable depression of the *latero-anterior* part of the chest gradually took place during the progress of recovery. As far as physical and symptomatic evidence can decide the point, there was certainly neither notable pleural exudation nor liquid effusion in this instance; but, as in Dr. Stokes's cases, there was no *post-mortem* examination to decide the question. I cannot help feeling persuaded that *simple* pneumonia may entail the alteration of shape which I thus believe I have actually witnessed. Perhaps, all things considered, the most efficient agent in producing depression of the chest after pleurisy, is the contraction of the plastic matter exuded on the pleural surfaces. Why should not the same contraction (occurring as a law of its existence) of exudation poured into the substance of the lung, cause *similar* alteration in the form of the thorax? I say similar, not the same in amount, because, in the case of pleurisy, there is another well-known cause of depression, which does not exist in the instance of pneumonia. It appears curious that M. Grisolle, who professes to have seen the size of the lung, enlarged by interstitial exudation solely, gradually return to its natural state, should maintain depression of the surface to be impossible. What is to prevent the tendency to diminution of bulk from gradually bringing the lung to a less volume than in health; and this once effected, will not depression of the parietes inevitably follow?

Such are the signs of pneumonia following the ordinary course; but it sometimes happens that, instead of interstitial or infiltrating suppuration, formation of *abscess* occurs. The signs are then peculiar. The student should remember that in diagnosticating pulmonary abscess, the first point, if possible, to be ascertained is, whether the signs of pneumonia have existed in the organ which is suspected to be the seat of purulent collection. Admitting this to be settled in the affirmative, the special signs of abscess will vary according as the pus has been more or less completely evacuated, or is still retained.

First, in the case of a *pulmonary abscess, of which the contents are more or less completely evacuated,* the diagnosis is grounded generally on the fact of the signs of excavation supervening upon those of pneumonia. The percussion-sound is dull, and the parietal resistance marked; or, in some cases, the resonance is tubular, amphoric, or cracked-metal; the respiration is cavernous, or tubular, accompanied with large-sized, thin mucous or cavernulous rhonchus. The vocal resonance may be bronchophonic, pectoriloquous, or null.

Secondly, in the case of *an abscess, with its contents retained,* in consequence of want of ready bronchial communication, the signs are, of necessity, extremely obscure. There are none, indeed, of an absolutely distinctive kind. The dulness is marked, the percussion-sound may be tubular, the respiration tubular, and the vocal resonance strongly bronchophonic. But all this might have existed before the formation of abscess.

Sphacelus of the lung,—one of the very rarest terminations of acute pneumonia,*—would be signified by the signs of a cavity; the expectoration growing, at the same time, profuse, and of peculiar fœtor.

An œdematous state of the parenchyma may remain long after the resolution of pneumonia. The signs of this state are elsewhere described.

II. *Symptoms.*—A fair proportion of adults suffer from *prodromata* in the shape of general febrile disturbance, of from one to four days' duration, before any local symptom of the disease makes its appearance. In the majority of cases the invasion is sudden.

The *invasion* of the disease is marked by rigors, followed by pain in the side, short cough, oppressed breathing, and sometimes cephalalgia and vomiting. Rigors are very rarely deficient; in a great proportion of cases, they form the phenomenon of invasion, with greater frequency, indeed, than in any affections, except ague, and perhaps puerperal fever. The respiration is often accelerated greatly out of proportion with the pulse, at the very outset,—before any local symptom or sign points to pneumonia.

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* The rarity with which gangrene of the lung originates in acute sthenic inflammation is now generally recognised. Of 305 cases of pneumonia analysed by M. Grisolle, not one terminated by gangrene; and of 70 cases in various journals perused by him, 5 only could be considered positive instances of this mode of termination of the acute disease.

Among the symptoms of the *actual disease* stands *pain in the side* (29 only of 301 patients escaped it; and in 161 of 182 it appeared within the first twelve hours: Grisolle); generally seated below the nipple on the affected side; rarely corresponding with the seat of the pneumonia; depending on co-existing pleuritic irritation, except in infinitely rare cases, where the lung substance itself appears painful; in the latter case, always slight; in the former, variable in amount; and increased by cough and parietal pressure. *Dyspnœa*, of variable amount, is a constant symptom; occurs within the first few hours; and raises the number of respirations to from 24 up to 80 per minute,—from 30 to 50 being the more usual extremes. When the respiration reaches 70 or 80, suffocation seems threatened, and speech is obstructed; 30 or 40 respirations per minute may exist without the patient being conscious of particular dyspnœa. Their frequency does not depend on any particular seat of the pneumonia, nor even, singularly enough, on its extent,—at least necessarily. I have known double pneumonia attended with a less number of respirations than inflammation of a limited portion of one lung. Although marked acceleration of breathing is an unfavourable sign, it is not, even to the highest degree, of fatal augury: recovery may take place where the respiration has reached 80 per minute. Now the circulation does not increase in frequency in the same proportion as the respiration,—hence the ratio of the two becomes more or less perverted. Thus, for 100 pulsations per minute, there may be 60 respirations, (I have seen this)—numbers giving a ratio of 1·7 : 1, instead of 4·5 : 1, that of health. The same perversion exists in those exceptional cases of pneumonia in which the pulse maintains a low frequency throughout. This perverted pulse-respiration ratio may, as I have found in several instances, *prove the first sign of pneumonia, appearing before crepitation or rusty expectoration;* as, *per contra*, a return to, or towards, the healthy standard, *may announce resolution some days earlier than the rhonchus crepitans redux.*[*] *Cough*, in the great majority of cases, occurs within the first twelve hours, moderate in amount, rarely paroxysmal, more severe in double than single pneumonia, and diminishing, occasionally even ceasing, towards the close in fatal cases. It is accompanied, in the majority of cases, with *expectoration* of absolutely pathognomonic character,—sanguinolent, or rusty in tint, viscid, semi-transparent, adhering to the vessel, but slightly aërated, passing through various shades of orange, apricot colour, faintly greenish, and lastly becoming white—then opaque and bronchial. In some cases the sputa are diffluent, watery, dull brown, or even faintly blackish,—resembling liquorice juice, or prune juice,—conditions of evil augury. The red tint of the sputa in the mass of cases comes on during the first forty-eight hours, and after the fourth day its appearance becomes very rare; the rusty tint may last from one to fourteen days. In some rare cases, the expectoration continues white throughout; and in yet rarer instances (especially where the pneumonia is central, or of the upper lobe,) there is no expectoration. If the expectoration in pneumonia be actually bloody—if there be hæmoptysis, in short—the pneumonia is tuberculous, according to my experience. The *expired air* is sensibly colder than natural, especially when the respiration is very frequent; and the quantity of *carbonic acid* expired is, according to the experiments of Nysten, diminished. It would also appear that accumulation of fat occurs to a great amount in the inflamed parenchyma;[†] doubtless, from the impermeability of the lung, products, which should be oxidised, fail to undergo that change. The well-known fact, that pneumonia does not cause such rapid emaciation as many other equally dangerous diseases, deserves attention in connection with this peculiarity.

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[*] *E. g.* Case of Craddock, Clin. Lect., loc. cit., p. 144.

[†] The natural proportion of fat to parenchyma being 10 per 100, it may rise to 50 per 100, according to N. Guillot.

Among the *general symptoms*, the state of the *pulse* is important. It may reach 140 or 160 beats per minute,—and in the mass of serious and fatal cases, is of much greater frequency than in those of recovery, generally attaining its maximum frequency by the third or fourth day. I have, however, known pneumonia run its course, both in old persons and in young adults, with a pulse never exceeding 60; in these cases, however, the healthy standard has, on recovery, proved still lower. Unless when very frequent, the pulse is habitually full and resisting: when feeble and small, this sometimes depends on embarrassed circulation, produced by *distension of the right cavities of the heart* with blood. Fulness of the jugular vein sometimes appears under such circumstances,—possibly, sometimes, from actual pressure on the vena cava by the hepatized lung: jugular pulsation occasionally occurs, and pulsation even of the dorsal veins of the hand has, in rare instances, been seen. The *blood*, buffed and cupped, is hyperinotic in the highest degree,—the fibrine reaching from 6 to 13 per 1000. There is a peculiar tendency to the formation of solid resistant coagula in the right heart, and in the pulmonary arteries, an obvious source of embarrassed circulation and other dangers; but I cannot agree with M. Bouillaud that their formation is an *invariable* attendant on hepatization. The temperature of the *skin* is raised; its feel often pungently, acridly, burningly hot, not more so in the vicinity of the inflamed part than over the rest of the chest. Sweating, sometimes copious, may occur towards the decline of the disease; and sudamina may form in abundance. The *digestive organs* sympathise. The *tongue* varies in appearance, but does not exhibit the adynamic (" typhoid ") character; *thirst* (scarcely in the ratio of the fever), *anorexia*, occasionally vomiting, tendency to constipation, or sometimes (in fatal cases towards the close especially) diarrhœa. Jaundice occasionally occurs, much more frequently when the right lung is affected than the left, but with little more frequency when the disease is seated in the lower rather than the upper lobe of that lung. The *urine* is febrile (deep-coloured, and of high specific gravity,) and often contains temporarily a small quantity of albumen, and towards convalescence deposits crystals of oxalate of lime, or triple phosphate, or lithates in abundance. Among *cerebral symptoms*, the only one of frequent occurrence is cephalalgia; it comes on with the outset, as part of the febrile state. Delirium, coma, and convulsions are rare; complete insomnia is seldom observed; the *organs of sense* are not specially affected; epistasis, however, is sometimes met with. *Prostration of strength*, as a rule, occurs from the first, and is so positive and so marked, that the fact may be made available in diagnosis: the exceptions are very rare. The *face*, more or less anxious in its expression, is of heightened colour,—the tint being actually red or tending to lividity,—or, especially when suppuration occurs, pale, yellowish, earthy-looking. The *decumbency* is most commonly dorsal, with slight inclination to one or the other side. Andral affirms that not one out of fifteen patients lies on the affected side.

III. The terminations of acute pneumonia are—(*a*) by resolution, (*b*) diffuse suppuration, (*c*) abscess, (*d*) gangrene, (*e*) lapse into the chronic state.

(*a*) Resolution, of which the signs have already been systematically set down, occurs at very various periods, and with different combinations of those signs. Thus, of one hundred and three convalescents, observed by M. Grisolle, and discharged from hospital between the twentieth and fifty-fifth days of the disease, thirty-seven had no morbid signs, thirty-six weak respiration, fourteen slightly blowing respiration, eleven redux crepitant or sub-crepitant rhonchus, and five deficient expansion with bronchitic rhonchi. The doctrine of crises and of critical days receives but little support from the phenomenon of pneumonia.*

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* It is just to state here, however, that Andral refers to 112 cases; of

It is most important to observe that the physical signs of resolution should, when the lung is affected throughout, make their appearance first at the apex. If they pursue the contrary course, travelling from below upwards, the existence of tubercle superiorly is to be strongly apprehended.

(*b*) The symptoms of diffuse suppuration are vague and unsatisfactory. Shivering may be completely absent, and dark fluid liquorice-juice expectoration may exist in the stage of red hepatization. The general symptoms become more severe, and of adynamic character,—dry tongue, sordes on the teeth, pinched features, anxious expression, clammy skin, failure of strength of pulse, wandering delirium, or somnolence, and semi-coma occur. But all this *may* happen in cases where no suppuration has taken place. Hence it follows, that the difficulty of proving the fact of recovery after diffuse suppuration, is extreme; in truth, there is no positive evidence of recovery having occurred in such cases.

(*c*) There are no positive symptomatic evidences of the formation of abscess. The contents may be fœtid, from a sloughing condition of the walls. Such a case is readily to be confounded with primary gangrene of the lung. Abscesses may terminate favourably by passing into the state of quiescent cavity; and it is alleged (although they early—sometimes in a week—become lined with a pyogenic, and, eventually, pseudo-mucous membrane) by perfect cicatrisation.

(*d, e.*) The symptoms of gangrene of the lung will be separately considered presently, as likewise the subject of chronic pneumonia.

IV. The *diagnosis* of acute pneumomia, as a rule, is simple. Its crepitant rhonchus, tubular breathing, and rusty expectoration are, when well marked, each and all peculiar to itself. The crepitant rhonchus is distinguished from the sub-crepitant of capillary bronchitis by its more distinct limitation to inspiration,

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which it is affirmed one-half terminated on the seventh, fourteenth, or twenty-first days.

its greater fineness, dryness, suddenness of evolution, and abundance. It exists at one base only,—the sub-crepitant at both; slight dulness under percussion soon attends true crepitation, and the signs of hepatization quickly ensue; the pulse-respiration ratio is seriously perverted, while in capillary bronchitis it suffers but slightly. *Œdema* of the lungs is distinguished by the comparatively large size and bubbling character of its rhonchus, by the absence of tubular breathing, by the absence of febrile action, heat of skin, and perverted ratio of the pulse and respiration, and by the circumstances under which the disease originates. Attention to the character of the different *pseudo-rhonchi* (pleural, mediastinal, and parenchymatous), as elsewhere described, will prevent their being mistaken for true crepitant rhonchus.

The distinctive marks of pneumonic solidification from a collection of fluid in the pleura, have already been given.

V. Pneumonia stands high among those diseases of which the fitting *treatment* has been established by scientific experience. First, venesection has been numerically proved to diminish the mortality and lessen the mean duration of the disease, and also curtail the duration of its prominent symptoms, both subjective and objective; the pain in the side, the febrile action, the peculiar expectoration, and the physical signs. Whether venesection have the power of actually arresting the disease at the very outset, and preventing the occurrence of hepatization, I hold to be yet scientifically undetermined; but that, in the immense majority of cases, it is vain to push bleeding to extremes in the hope of producing any such effect, clinical observation daily proves. In acute sthenic pneumonia, there are few barriers to venesection. Advanced age is none: Morgagni bled nonagenarians with success. Neither do pregnancy nor menstruation, provided the indications be otherwise positive, stand in the way of the use of the lancet. I have repeatedly bled women with thoracic inflammation during the flow of the catamenia, without arresting the discharge; and if such stoppage should occur, cupping over the sacrum, or leeching the perinæum, will prevent any ill consequences. During certain epidemics, bleeding is very badly borne; and persons of a constitution shattered by excess, social anxiety, physical privation, or chronic disease, should, of course, be cautiously deprived of any of their, already spanæmic or hypinotic, blood. The earlier the bleeding, the better. M. Louis has shown that pneumonic patients, bled within the first four days, recover, *cæteris paribus*, four or five days sooner than those bled at a more advanced period; and Dr. Jackson, the enlightened practitioner of Boston, has proved that by bleeding on the first day, the mean duration, in a mass of cases at the Massachusetts Hospital, was lowered from 14·60 to 11 days.* No *period* of the disease is too late for blood-letting, provided the indication be thoroughly and strongly established on general principles. Even the *stage* of suppuration is by some held not to be a contra-indication, in itself alone, to the use of the lancet; but, although the name of M. Andral appears among those of the supporters of this doctrine, I have the strongest doubts of its correctness. M. Grisolle refers to four patients, bled to ten or twelve ounces, and in whom *post-mortem* examination (the sole positive test) proved the existence of the purulent stage. In all four cases, the fatal issue was obviously hastened (in one almost immediately caused) by the loss of blood.† No fixed rule can be laid down for the quantity of blood to be drawn; the mean amount of four pounds five ounces, taken from his patients by M. Bouillaud, has been most satisfactorily proved by M. Grisolle to have produced no more favourable immediate results (and of the ultimate ones what may not be feared?) than the abstraction of a mean quantity of two pounds seven ounces from a mass of patients treated by himself and others.* For my own part, I strongly question the utility of even such amount of depletion as this. Certainly, few cases have presented themselves to me in London practice, where it was necessary to draw blood oftener than twice; sixteen ounces sufficing in the first instance, and some ten or twelve in the second. Slow convalescence is not the worst evil in cases where blood has been too lavishly sacrificed: a form of spanæmia is sometimes induced, which it may take months, nay years, to recover from.

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* Appendix to Putnam's translation of Louis on the Effects of Blood-letting. Boston, U. S., 1836.

† There is an anatomical fact but little known, which may have some important bearing on the question of bleeding in the first stage. I mean the fact, that sometimes a lung apparently in a state of suppuration is in reality infiltrated with softened fibrinous exudation,—exudation cells alone, and no pus-cells, being found with the microscope. But who shall distinguish, during life, the case of softened exudation from that of suppuration?

Leeching, or rather cupping, over the affected part, should always be employed in addition (in very mild cases it will suffice alone) to general bleeding: local abstraction of blood affects pain much more directly and quickly than venesection. Six or eight ounces may, with propriety, be taken in a case of medium intensity (in addition to the quantity taken from a vein) by cupping: all local pain sometimes instantly disappears after the operation.

Tartarised antimony stands next in importance to blood-letting in the treatment of pneumonia,—were I, indeed, henceforth, in the management of this disease, forced to surrender either, on the one hand, venesection, or, on the other, cupping and tartarised antimony, I should not hesitate to relinquish the former. In what manner this important agent produces its beneficial effects on the lung, is matter of the loosest speculation;—that it does produce such effects, is the really important point, and one of which scientific proofs abound. There is not any available evidence to show positively whether the effects of antimony on pneumonia are more marked when the mineral is (as it is technically called) *tolerated* perfectly or imperfectly, or when it is not *tolerated* at all. The question could obviously only be decided by numerical comparison; and the number of cases in which complete tolerance is observed (that is, total absence of effects on the stomach and bowels) is relatively very small. Improvement often takes place within eight or ten hours after the medicine has been commenced with, and without any notable effect on the alimentary canal being noticed; whereas recovery also ensues when it acts freely both as an emetic and purgative. Hence it is more as a result of prejudice (for what but prejudices are even plausible *à priori* theories?) than of logical deduction from experience, that, in imitation of Rasori and Laennec, I prescribe antimony in such manner and combinations as are most likely to prevent its disturbing the stomach. The salt should at first be given in doses of half a grain, combined with dilute hydrocyanic acid, paregoric, and tincture of orange-peel, every hour for the first three or four hours,—and the dose then increased, at intervals of two hours, to one grain: in the course of twelve hours the quantity may be raised to two grains,—its repetition made less frequent, say every fourth hour.

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* Even in our own country, it was at one time thought by many that bleeding could scarcely be pushed far enough: men were systematically bled to convulsions. It was held theoretically sound to take away blood, the source of the existing evil, to the uttermost point; but it was forgotten, or it was not known, that the increased impetus of the circulation during hæmorrhagic reaction might make up for the diminished quantity propelled. In those days, too, provided *theory* were satisfied, *facts* were held as matters of no importance. "Dr. Gregory, of Edinburgh," reports Dr. Watson, "used to bleed to the verge of convulsion. His colleague, Dr. Rutherford, seldom went beyond three bleedings, and generally accomplished his object by two. *His* patients recovered quickly; *Dr. Gregory's* very slowly." Yet Dr. Gregory continued to cling to his practice; for he had theory on his side.

The constitutional effect of mercury is by some held to be peculiarly efficacious in the stage of red hepatization. It is even maintained that when that stage has been reached, calomel is a more valuable medicine than antimony. No scientific demonstration of this view exists. If it were correct, the value of antimony in hospital practice, at least, would be singularly small; for the great majority of persons, admitted into hospitals, have some amount of hepatization, when first seen. Mercurials appear to me to be desirable in those cases of pneumonia only, where, for some cause or other, antimony is inadmissible. It seems a point worth submitting, under proper conditions, to the test of experience, whether the free and rapid administration of alkalies might not be useful in pneumonia, attended, as it is, with the maximum amount of hyperinosis observed in any disease.

Blisters are not advisable in the earliest periods of pneumonia: it would appear that they have no effect in shortening the mean duration of the disease, and they certainly increase fever and general irritation at the outset of the attack. At its more advanced periods, when fever has been materially controlled, they certainly relieve pain and dyspnœa, and seem to promote absorption.

The ordinary *juvantia* of the antiphlogistic regimen must, of course, be carefully put in requisition; the bowels, if necessary, should be opened by medicine; but profuse purgation is, to say the least, absolutely useless.

Complete demonstration of the utility of treatment in pneumonia is found in the fact that the mortality of the disease steadily increases with each succeeding day it has been allowed to run its course uncontrolled. The statistics of M. Grisolle (referring to the treatment by moderate bleeding and tartar emetic) show, that while the mortality among those first seen and treated within the two first days, is only one-thirteenth, it rises among those whose treatment does not commence till the eighth day, from one-third to one-half of the whole number. But, on the other hand, it is important to remember that there are certain conditions beyond the control of the physician, which exercise a most indubitable influence on the issue of the disease. Of all these, age is the most important: while at the two extremes of life (the new-born infant and the octogenarian) the disease is almost inevitably fatal; the mortality between the ages of six and twelve scarcely exceeds two-and-a-half per cent. Between the ages of fifteen and thirty, the deaths equal about six per cent. of those attacked; suddenly rise to about fourteen per cent. in persons aged between thirty and forty; and thenceforth steadily increase with each succeeding decade.

Hence it follows, that in estimating the value of any system of treatment, the age of the pneumonic patients treated is an element of primary importance. There are periods of life at which it is next to impossible to save—there are periods of life at which it is not easy, with common prudence, to lose—a sufferer from idiopathic and sthenic pneumonia.

§ II. *Chronic Pneumonia* is rare as a sequence of the acute disease; it is rare as a primary disease; it is common as a local attendant on the progress of tubercle, cancer, and other adventitious products in the lung. I mean by chronic pneumonia, that form of disease in which an impermeable tissue is infiltrated with *toughly-solid* exudation (in the state of induration-matter), and where there is no tendency to a softening process: these are its *main* characters.

I. When acute pneumonia lapses into the chronic disease, the strength and flesh, instead of returning with more or less rapidity, continue to fail; there is habitual, though moderate, dyspnœa; sensations of discomfort and oppression within the chest are almost constantly present; cough, with insignificant expectoration, and no hemoptysis, exists; there are thirst and anorexia, with irregular fever, which gradually grows constant,—has its evening-exacerbation, but rarely any notable night-perspirations: with all this, the loss of flesh may, for a time, almost equal that occurring in the same period in phthisis.

II. Physical signs mark the changes in the lung: the surface is more or less extensively depressed, according to the area implicated; the chest movements are impaired, especially the costal ones; the antero-posterior diameter, and the superficial width of the side are diminished; and the percussion-sound is dull (sometimes wooden or tubular), with marked parietal resistance. The respiration is weak, uneven in quantity, harsh, bronchial, or diffused blowing in the affected parts,—occasionally exaggerated beyond these. The vocal resonance varies; it may be bronchophonic or null: the vocal fremitus is intensified. Chronic pneumonia has no rhonchus of its own; but there may be subcrepitation from bronchitis or œdema. In all probability, under favourable circumstances, interstitial creaking-sound may be produced by forced inspiration in lung-substance of this kind.

M. Grisolle refers to a case observed by M. Requin, which shows that the physical signs in chronic consolidation may be of a very different kind,—in fact, all of them negative: total absence of all healthy or morbid respiratory murmurs, of rhonchus, and vocal resonance, the percussion-sound at the time being completely dull. In the case referred to, the affection was mistaken for simple pleuritic effusion; but the patient dying *in a state of marasmus* two or three months after the outset of the affection, the sole morbid condition discovered in the chest was very firm (neither granular nor tuberculous) induration of the lower lobe of the right lung. This is a parallel state of things to that sometimes observed in acute solidification.

III. In cases where the affection principally implicates the upper lobe, and where the contraction of the exudation-matter thrown out into the substance of the lung has been active, flattening of the infra-clavicular region will take place. Under these circumstances, especially if, as often is the fact, among the general symptoms appear emaciation, slow fever, &c., the distinction of the case from tuberculous consolidation is extremely difficult—impossible, indeed, unless by the aid of repeated examinations at certain intervals of time. The comparatively stationary condition of the part in simple consolidation, associated with the progress of the general symptoms, if it do not perfectly explain the nature of the case, will, at least, point to the necessity of a cautious diagnosis. Fortunately difficulties of the class now especially referred to are of singularly rare occurrence.

IV. The treatment of chronic pneumonia is not essentially different from that of the early stages of tuberculisation. If the diagnosis were positively established, the occasional application of a few leeches, or the abstraction of three or four ounces of blood, might be more freely ventured upon, in the absence of all acute symptoms, than in phthisis.

§ III. The *varieties* of pneumonia are very numerous, and referrible to the *seat*, *course*, and *primary* or *secondary* character of the disease.

I. (*a*) The seat of pneumonia, though mainly interesting anatomically, is not devoid of clinical import, as a guide to the observer in quest of the physical signs of the disease. Of one thousand four hundred and thirty cases, seven hundred and forty-two were of the right lung, four hundred and twenty-six of the left, and two hundred and sixty-two of both organs. In two hundred and sixty-four cases, the upper lobe was affected one hundred and one, the lower one hundred and thirty-three, the middle part thirty, times (Grisolle). With respect to the cases of double pneumonia, which hold a rather high numerical rank (they furnish 18·3 per 100 of the whole series), it is to be observed that the great majority of them were not so from the outset; in other words, that the implication of the second lung was secondary in point of time. This, indeed, is a matter of no mean importance; for in doubtful cases the existence of the phenomenon at one only or at both sides of the chest, will aid materially in distinguishing the true crepitant rhonchus of pneumonia from the subcrepitant of capillary bronchitis. And even with the qualification now mentioned, alone, the frequency of double pneumonia is probably considerably exaggerated in the estimate just given: subcrepitation has often been mistaken for true crepitation, and a double capillary bronchitis put down as a double pneumonia; it is traditionally well known in Paris that even Laennec committed this error. The age of patients, too, must be borne in mind: in the adult, the proportion of double pneumonias does not probably much exceed one in twelve;* it has even been estimated so low as one in seventeen. On the other hand, the disease is almost always double in newborn infants; in one hundred and twenty-eight such cases observed by M.M. Valleix and Vernois, the right lung alone suffered in seventeen cases, the left alone in no single instance; while both lungs were affected one hundred and seventeen times. Pneumonia commencing about the middle of the lung is rarely primary: it is commonly either a sequence of endo-pericarditis or of blood origin,—an anatomical fact of obvious practical signification. The anterior *margin* of one or both lungs is sometimes separately inflamed: I believe that the frequency of this peculiar seat has been exaggerated from confounding mediastinal pseudo-crepitation with true pneumonic rhonchus.

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* In the scholar year 1834-5, when I was attending at the Hôtel-Dieu, 48 cases of pneumonia occurred in the wards of M. Chomel: 33 of these were of the right lung; 11 of the left; 4 were double.

(*b*) The pneumonia of infancy very frequently (but by no means so constantly as is usually taught), instead of spreading through a lobe of the lung generally, limits itself to scattered groups of lobules, the intervening tissue remaining sound: such pneumonia is called *lobular.** So, too, pneumonia preceding the formation of secondary abscesses in the lungs, sequential to phlebitis, &c., commonly assumes this form, no matter what be the age of the individual.

The physical signs of lobular pneumonia are obscure. Inspection, application of the hand, and mensuration give merely negative results. Percussion, too, does not disclose such an amount of dulness as can be clinically trusted to; which is no more than might be anticipated, when we consider that the nodules of consolidated lung are separated by tissue perfectly permeable. In many cases originally (to all appearance) lobular, I have found the sound duller than natural, it is true; but when this was the case, and the opportunity of examining the parts occurred, I invariably discovered such extension of the inflammation between the nodules as to reduce the organ, physically speaking, almost to the state of ordinary consolidation. The respiration is exaggerated in some points; harsh, bronchial, or even slightly blowing (never tubular, so long as the pneumonia is simply lobular,) in the spots probably corresponding to the consolidated nodules. Occasionally a few cracklings of an imperfect crepitant rhonchus may be herad; but it is difficult to distinguish these from the humid rhonchus of fine bronchitis,—a disease almost constantly associated in children with inflammation of the parenchyma.*

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* True lobular pneumonia is distinguished in the dead subject from collapse, and consequent solidity, of lobules, by its being insusceptible of inflation; while merely collapsed nodules of tissue, as originally shown by Bailly and Legendre, may be blown up to their natural, or very nearly their natural, state. It is impossible to say in how many of the series of infantile pneumonias, above referred to, the real condition was nothing more than the collapse in question.

(*c*) The *interlobular cellular tissue* may be the seat of acute suppurative inflammation,† pus occupying the situation that is filled by air in interlobular emphysema. Or this tissue may be infiltrated with fibrinous exudation, which solidifies into induration-matter, and causes considerable contraction of the lung, and sinking in of the side (Corrigan's "cirrhosis"). The bronchi undergo marked dilatation; and the pulmonary tissue, compressed by these tubes and by the surrounding induration-matter, acting with its peculiar steady and constant force, becomes almost completely impermeable.‡

The physical signs are flattening and diminished width of the side, impaired costal motion, increased vocal fremitus, percussion hard and dull or tubular, respiration irregular in distribution, weak, deep-seated, bronchial, or diffused blowing; while the rhonchi of bronchitis with hollow respiration indicate the existence of dilated tubes. The heart may, by this condition of things, be drawn to the affected side: as, however, there is very generally co-existent agglutination of the two laminæ of the pleura, it is difficult to say to what extent the state of the lung alone contributes to the displacement of the heart; such was the fact in the case of S. Osmond, referred to in the note in the preceding page.

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* The signs of *diffuse* pneumonia in the infant scarcely differ from those noticed in the adult. Crepitation, metallic tubular breathing, and dulness under percussion are the essential signs; the child's cry resounds with sniffling and bronchophonic character. The crepitation is of larger size than in the adult.

† Carswell's framed drawings, U. C. Museum, No. 57, C. b. 573.

‡ In a remarkable case of the kind (S. Osmond, U. C. H., Males, vol. iv., p. 336,) I found an infiltrated cellulo-fibrous tissue actually replacing certain lobules of lung: the pulmonary texture had been absorbed.

The distinction during life of this state of the lung from simple chronic pneumonia is always difficult, sometimes impossible. In "cirrhosis" of the organ, retraction of the side is, however, greatly more marked than in ordinary chronic inflammation; and if there be a considerable amount of flattening, we may be certain that it is not caused by the latter disease alone. The tubular percussion-sound, stronger respiration, signs of dilated bronchi, and traction of the heart towards the affected side (only distinguishable on the right), met with in cirrhosis, are not observed in the simple disease.

II. (*a*) Instead of running its ordinary course with marked subjective symptoms, pneumonia may be completely *latent.* The perverted ratio of the pulse and respiration, and the physical signs, are then the sole guides to the detection of the disease. Pneumonia occurs in this form solely under. circumstances of general physical debility: it is either *senile* or connected with *adynamic* diseases, of which it is an intercurrent phenomenon.

Physically, latent pneumonia is characterised by the rapidity with which it runs into solidification, and with which it involves a great extent of substance.

In managing this form of inflammation, the main attention must be given to the state of the system generally. Venesection I cannot believe to be ever requisite; and abstraction of blood, even locally by cupping, should be very cautiously ventured on. Still, if the respiration be much accelerated, and consolidation very rapidly extending, a few ounces of blood may be taken by cupping. Dry-cupping is always a measure of utility, and unattended with danger. The early application of blisters is by some observers strongly recommended in this variety of the disease: I have not happened to observe results justifying their confidence. Sesqui-carbonate of ammonia, bark, and wine—the remedies for the existing state of system at large—exercise, in my opinion, the most obvious and immediate good effects on the local disease.

(*b*) Under the phrase "*hypostatic* pneumonia" have been described mere passive congestion of the bases of the lungs, occurring shortly before death, and also the senile and adynamic inflammations just referred to.

Where the tendency to such congestion exhibits itself, occasional change of posture from the back to the sides, or even to the prone position, is one of the most important remedies; and, indeed, this is true, more or less, of every variety of pneumonia.

III. It is impossible to exaggerate the importance of pneumonia in its next variety,—namely, when occurring as a *secondary* or *intercurrent* disease: in truth, the majority of cases of pneumonia belong to this class. It is intercurrent pneumonia that commonly kills new-born infants, affected with hardening of the cellular tissue and diphtheritic disease. From childhood to puberty, croup, cancrum oris, measles, hooping-cough, variola, frequently prove fatal (especially croup), through inflammation of the lungs. Again, we meet it complicating the diseases of the adult,—and if not at this period so frequently fatal, not the less important for the practitioner to watch. Thus it appears in typhoid fever, phlebitis, glanders, puerperal fever, inflammation of the bowels, and of the brain or membranes, and in acute rheumatism; among chronic diseases, in pulmonary tubercle, Bright's disease, chronic affections of the liver, not so commonly as might be expected in organic diseases of the heart, in cancerous affections, not only of the thoracic, but of distant organs, &c.

In treating intercurrent pneumonia, we must remember that the inflammatory character of the local malady is modified more or less seriously by the general state of the system. It is exceedingly probable, indeed, that various differences exist in the intimate constitution of many of the intercurrent pneumonias,—though at present no absolute proof of the fact can be given. Hence, if antiphlogistic management be proper, as it positively is in these cases, the state of the system at large should always be allowed full control. This is more especially true in the instance of diathetic diseases, such as rheumatism: it may be that colchicum is a more important remedy than antimony for rheumatic pneumonia. In pneumonia complicating purpura, the treatment (except in regard of blistering and dry-cupping) is wholly that of the blood-disease present.

GANGRENE OF THE LUNG.

I. Gangrene of the lung occurs in two anatomical forms,—the diffused and the circumscribed;—the latter, greatly more frequent than the former, is distinguished by the sharp line of demarcation between the gangrenous and sound tissue.

II. (*a*) The symptoms in the *diffused* form are great general prostration, suppressed breathing, profuse expectoration, frothy and purulent-looking, of gangrenous odour, with a small, feeble, and very frequent pulse, and all the general appearances of intense adynamia. The power to expectorate is soon lost, and death occurs from suffocation.

(*b*) The course of *circumscribed* gangrene is somewhat different. At first the evidences of affection of the lung are commonly extremely obscure; the signs of pulmonary congestion exist, coupled with an amount of prostration quite out of proportion with the extent of local disease; the expectoration muco-purulent, rarely bloody in adults, frequently so in infants and children, acquires, the moment communication is established between the bronchial tubes and the gangrenous tissue, properties more or less strongly characteristic of the disease. It is of dirty-greenish, yellowish-brown, or ash-grey colour; very liquid; and exhales an odour distinctly gangrenous, or resembling that of wet plaster, or *sui generis* but painfully fœtid. In the adult the breath generally has the same fœtor, but this may not be constant: the expired air may be completely free for some minutes at a time from disagreeable smell; when suddenly, without cough or any other apparent cause, it becomes intolerably fœtid. Possibly temporary plugging of the bronchi communicating with the mortified tissue may account for the absence of odour. In children, fœtor of the breath is less common than in adults.

The terminations of circumscribed gangrene are by death or recovery. Death may occur rapidly by collapse, or, in children, sometimes by hæmoptysis. Or the fatal event may take place slowly: abundant purulent fœtid expectoration, hectic fever, night sweats, emaciation, wear out the patient, and, after protracted suffering of weeks, or even months, death occurs. In cases of recovery (unfortunately a small minority), the discharge loses its fœtor, diminishes in quantity, and becomes simply mucous; the hectic fever ceases, the appetite returns, and a complete rally is gradually accomplished.

The diagnosis of gangrene of the lung turns upon the peculiar fœtor of the breath and expectoration, coupled with the physical signs of softening and excavation of the substance of the lung, ensuing upon those of passive congestion or sub-acute adynamic pneumonia. But there are sources of fallacy of two kinds; there may be the extremest fœtor and profuse greenish sero-purulent expectoration, and yet no true gangrenous cavity formed; and there may be a gangrenous cavity without the characteristic conditions of the breath and sputa.

In the first class of cases appear examples of acute added to chronic bronchitis, where the breath and sputa acquire gangrenous odour, without any evidence of excavation,—where recovery takes place, the fœtor being merely temporary, and where the only plausible explanation of the facts is, that sloughing of the mucous membrane has occurred within the tubes on a small scale. I have no proof of this view, however, to offer in the form of *post-mortem* examination. Again, the breath and sputa sometimes acquire gangrenous odour, in cases of broncho pleural fistula or empyema, where changes in the pleural sac are the real causes of the fœtor. But even the positive signs of cavity in the lung *plus* the fœtor, do not prove that the cavity is of gangrenous character. It may be tuberculous, or it may be purulent. I have now seen some half-dozen cases of consumption in which the special fœtor occurred incidentally in connection with tuberculous cavities already formed. In one of these instances (Consumption Hospital, Chelsea), the expectoration of a fœtid pea-like mass, distinctly possessing microscopically, and even to the naked eye, the characters of pulmonary tissue, put a term to the gangrenous discharge,—a fact which I hold to be proof positive that a minute sphacelated spot may impress the characteristic fœtor upon the expectoration and breath, quite as effectually as gangrene of extensive area. A tuberculous cavity, thus locally gangrenous, is very difficult to distinguish from true gangrene of the lung, if the case be seen for the first time when that change has occurred, and if the history of the case be imperfect. The seat of the cavity at the apex, and the existing signs of induration at the other upper lobe, once guided me successfully to the diagnosis in a case of this kind; but I am far from thinking the observer would always be justified in an absolute affirmation on such data. A fœtid abscess is generally distinguishable from true gangrene, not by the amount of fœtor, which may be just as great from local sloughing of the walls of the abscess, but by the fact that the signs of excavation precede the occurrence of fœtor in the case of abscess, follow it in that of true gangrene.*

The second variety of case, where real gangrene occurs without perceptible fœtor, is rare, especially in the adult. In children a guide to the diagnosis of an excavation thus formed, is sometimes found in hæmoptysis; for, singularly enough, while tubercle scarcely ever causes hæmoptysis in childhood, gangrene is then frequently attended with that symptom.

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* In a case which fell recently under my observation (Dl. Hayes, U. C. H., March, 1851), there were occasionally such abundant discharges of almost pure pus, alternating with the more characteristic diffluent gangrenous expectoration, that, had not fœtor preceded by some days the signs of softening and excavation, I should have been disposed to regard the case as one of fœtid abscesses successively bursting, and not of gangrene: unfortunately permission to examine the body could not be obtained.

III. In the *treatment* of gangrene of the lung, the chief reliance is to be placed, in the acute state, in stimulants and tonics: the sesqui-carbonate of ammonia, opium and camphor, in various combinations with bark, or quinine, given in full and repeated doses, certainly afford the patient the best chances of recovery. The effect on the powers of the system generally, often produced by the first few doses, is really extraordinary. Wine or brandy may be administered at the same time.

The only local measures advisable, under ordinary circumstances, are, dry-cupping and counter-irritation by blistering or otherwise. If, however, the evidences of acute secondary pneumonia produced by the irritative action of the sloughed lung, are conclusive, and the system generally has rallied, a few ounces of blood might be cautiously abstracted by leeches or cupping, from the affected side: the positive indication for this practice will, however, very rarely arise.

In cases lapsing into the chronic state, the mineral acids and quinine (of the former, the nitro-muriatic is probably the best) become the main remedies.

Fœtor should be corrected by chlorinated mouth-washes, or fluids containing creasote in suspension. The chloride of zinc may be used for this purpose in a state of extreme dilution (three grains to eight ounces of water). Inhalation of tar vapour and of chlorine should be had recourse to, not only as corrective of fœtor, but as tending, in all probability, by their direct chemical action on the sphacelated tissue, to control septic changes within the lung, and so lessen the local irritation and constitutional depression.

The diet should be nutritious and digestible: strong beef-tea, thickened with isinglass or prepared gelatine, finely-pounded meat, eggs beaten up with small quantities of brandy, milk, &c.,

should be given as frequently as the digestive powers of the patient appear to permit. Porter (or, better, stout) in moderation is an advisable beverage.

HÆMORRHAGE AND HÆMOPTYSIS.

§ I.—*Hæmorrhage* may occur from the mucous membrane, or other actual tissue, of the tubes; within the tissue of the lung itself; or into the cavity of the pleura: it is, accordingly, either *bronchial*, *pulmonary*, or *pleural*.

I.—It is generally stated in systematic works that *bronchial* hæmorrhage is extremely frequent; but, if the term be understood strictly in the sense above given it, I cannot help believing that it is of extraordinary rarity. Setting aside those instances—mere curiosities from their singularity—in which ulcers in the bronchial tubes furnish the blood of hæmoptysis, I have never yet seen a case where blood discharged during life even *appeared*, much less *was proved* (on inspection of the bronchial tubes) to have come from their substance by molecular ruptures (exhalation of the older writers). True, there is no *à priori* reason why blood should not ooze from the bronchial mucous membrane, as we know it does, as we actually sometimes see it do, from the mucous membrane of the mouth: but, on the one hand, the evidences of the occurrence are wanting; and, on the other, it is found in ninety-nine hundredths of cases, when blood has made its way into the bronchial tubes, that the pulmonary tissue itself is the source of supply. In cases of diseased heart attended with hæmoptysis during life, there may always, as far as I have examined, be found more or less marked evidence that the parenchyma of the lung, and not the mucous membrane, has given way molecularly: the evidence I refer to, is the presence of dark blood-points here and there in the pulmonary tissue—a sort of embryo pulmonary apoplexy. While, on the other hand, I have often found the finer tubes, as far as they can be followed with a scissors, free from undue vascularity or marks of saturation with blood,—a fact the more remarkable, because the larger trunks are occasionally, in such cases, very evidently imbibed with that fluid.

When blood *is* furnished by the bronchial tubes, hæmoptysis is its symptom, and thin bubbling abundant liquid rhonchus in the bronchi its physical sign. It does not appear to stagnate sufficiently within the tubes, when of this origin, to alter the percussion-sound.

II.—Pleural hæmorrhage, or *hæmothorax*, is in the majority of cases traumatic, from wounds, contusions, and fractures of the ribs. When non-traumatic, it is always a secondary state; for of pure idiopathic, passive or active, hæmorrhage into the pleural cavity, I have never heard of a positive example. The fluid of pleurisy contains blood in considerable quantities in some cases; but no difference is thereby impressed on the physical signs, or (notwithstanding the opinion of Laennec) perceptibly on the progress of the disease. In various blood-diseases with hæmorrhagic tendency, serous pleural effusion becomes stained with blood. Carcinoma of the lung bursting into the pleura, or carcinoma of the pleura itself undergoing superficial ruptures,* or apoplexy of the lung, making its way through the pulmonary pleura, or rupture of an aneurism, may all produce hæmothorax.

The physical signs of traumatic hæmothorax are dulness under percussion, enfeebled or annulled vocal fremitus, weak or suppressed respiration, absence of rhonchus, the vocal resonance being in a variable state, occasionally ægophonic,—signs coming on suddenly without inflammation and under circumstances of injury to the chest. In cases where extensive accumulation of blood occurs in the pleura from any of the medical diseases named, similar physical signs will be observed,—but they are rendered obscure and uncertain by the primary disease. In the instance of a ruptured aneurism, the constitutional symptoms of internal hæmorrhage will be observed.

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* Case of Dewing, U. C. H., Males, vol. v., p. 19.

III.—Pulmonary hæmorrhage, that is hæmorrhage springing from the actual parenchyma, occurs with or without recognised anatomical characters. In the first case we meet with the nodular and the uncircumscribed pulmonary apoplexy of Laennec, petechial, and, lastly, interlobular hæmorrhage,—an example of which hitherto undescribed form I once observed in a new-born infant. No one of these anatomical states is necessarily productive of, or connected with, hæmoptysis;* whereas there is no established morbid anatomy of the most frequent variety of hæmoptysis, that depending on consumption. Molecular ruptures of the capillary vessels of the parenchyma are doubtless the cause of the discharge of blood in tuberculous diseases, except in those infinitely rare cases where a vessel of some size is perforated; but absolute demonstration of the fact is still a desideratum.

Pulmonary apoplexy, in the nodular and uncircumscribed forms, is almost invariably an effect of disease of the heart, especially of the mitral orifice. It sometimes occurs, we are assured, from a "diseased state of the pulmonary vessels and parenchyma," independently of heart-disease. This I have never seen, except in cases (evidently not referred to in the above phrase) of cancer of the lung.

The symptoms of pulmonary apoplexy are exceedingly difficult to specify, because they are mixed up with those of pre-existing disease in the lungs. Dyspnœa, tightness, and dull pain in the chest, all exist independently of such apoplexy in mitral disease; they, however, increase in severity when blood escapes into the lung-substance, as does likewise any cough previously present. The only symptom really important is hæmoptysis, in the forms of tinged mucus,* striæ of blood, pure blood, rarely florid, rather darkish and dirty-looking. Profuse hæmoptysis is singularly rare; and there is no special appearance in the blood positively distinctive of its origin,—the character of most significance is the dark dirty aspect just mentioned.

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* It has been shown, indeed, by Dr. Watson, that nodular apoplexy may sometimes be an *effect*, instead of being the *cause*, of discharge of blood through the mouth. In a man dying from hæmorrhage from the lingual artery, several apoplectic nodules were found, evidently formed of blood within the air-cells, which had trickled downwards from the mouth through the windpipe.

The physical signs, too, are obscure. The effusion of blood is rarely (I have *never* seen it so) sufficiently great to act as an impediment to chest-motion. I have known the vocal vibration somewhat intensified over the nodular masses,† and the percussion rendered very notably dull. Over the accumulated blood the respiration is weak, or even suppressed; beyond it, harsh, bronchial, or diffused blowing; the state of vocal resonance varies. If there be hæmoptysis, thin, liquid rhonchi will be present. Even within the area of the apoplectic nodules, subcrepitant rhonchus may sometimes be caught on full inspiration. Instead of undergoing gradual absorption, pulmonary apoplexy may act as a source of irritation; the signs of local pneumonia, abscess, and, in rare cases, even of gangrene, may then be successively noted.

In regard of treatment, I am disposed to insist most strongly on the value of extensive and repeated dry-cupping. I have repeatedly known hæmoptysis stopped almost at once by this measure, in cases of mitral disease, when the general aspect of the patient forbade the abstraction of blood. A small quantity of blood may be removed with propriety, where there is no prominent asthenia. Counter-irritation, by blistering and otherwise, and free purgation, are the next most important remedies. The treatment must always be controlled by the state of the heart and of the secondary disorders dependent on that organ. Unless the hæmoptysis be considerable, it is not advisable to employ the ordinary astringents; digitalis will be useful or detrimental according to the condition of the heart's substance and orifices,—a matter to be discussed hereafter.

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* Blood-discs may often be found in the sputa of persons with mitral disease, where there is no blood visible to the naked eye.

† This is, however, a sign of slight value.

§ II.—Under the title of *Hæmoptysis*, or expectoration of blood, may be included all instances of discharge of that fluid, from any part of the air-passages below the epiglottis. The quantity of blood may be so small as to be barely visible to the naked eye, or it may amount to pints, or even quarts.

Hæmoptysis may occur, *mechanically*, from diminished pressure of the atmosphere, as in the ascent of lofty mountains, and from injuries of various kinds to the chest; and it may take place *vicariously*, as a periodical discharge in females, instead of the catamenia; unless, under these circumstances, it is a sign of *disease of the lungs, heart, or great vessels.* The diseases which act as more or less frequent causes of hæmoptysis, are tubercle, cancer, aneurism, cardiac diseases, and certain affections of the larynx, trachea, and bronchial tubes. In actual clinical practice, hæmoptysis is so frequently connected with tuberculous disease, that it comes to be one of its most significant symptoms. The laws of tuberculous hæmoptysis will hereafter be considered. In the present place, I shall simply place before the reader an analysis of the clinical evidence I have been able to collect on the question of the connection of hæmoptysis with tubercle *in adults.*

"(*a.*) The *quantity* of blood voided, is the first point for consideration. It is commonly said that the expectoration of streaked or tinged sputa is utterly insignificant, because such are seen in *bronchitis*, &c., &c.; but no attempt has ever been made to decide numerically to what extent this is true. I find that in 25 cases (observed at Brompton, and at University College Hospital) of chronic bronchitis with or without marked emphysema (*but always without serious disease of the heart*), the absence of such expectoration was noted in nineteen cases, its presence in six. Now in *all* these six cases of streaked expectoration, there was more or less ground for suspicion that tubercles were to a slight amount present,—in two of them this was proved to be the fact by *post-mortem* examination. While, then, as I have found, bloody expectoration occurs in 71·79 per cent. of tuberculised persons in the first stage, it occurred in 24 per cent, of bronchitic people (free from serious cardiac disease); but in all of the latter there was either suspicion or certainty of the existence of tubercle to a *slight* amount.* The mean duration of the disease in the phthisical cases was 26·55 months, in the bronchitic, 49·50,—hence the significance (*quoad* tubercle) of hæmoptysis is greater even than the relative per-centages above given would signify. Streaked or tinged sputa are rarely or never the 'first symptom' of phthisis; should they appear in this guise, then, they would probably be dependent on some other cause. The question of hæmoptysis in plastic bronchitis has already been referred to.

"(*b.*) *Primary cancer of the lung and mediastina*, as I have elsewhere shown, from the analysis of a small number of recorded cases, is very frequently attended with sanguineous expectoration or pure hæmoptysis. In regard of this symptom, the two diseases may be thus compared:—the per-centage of hæmoptysis *of all amounts* in cases of cancer is 72, in phthisis 80·92; while hæmoptysis *above one ounce* occurs in cancer and phthisis in the ratio of about 70 to 40. Hence 100 cases of cancer of the lung will be attended nearly as often with hæmoptysis of all amounts, and greatly more often with hæmoptysis above an ounce in amount at a time, than 100 cases of phthisis. But, on the other hand, tuberculous is so vastly more frequent than cancerous disease of the lung, that the share of the population suffering at any time from cancerous hæmoptysis will form but an insignificant fraction of that suffering from hæmoptysis of tuberculous origin.

"(*c.*) It is not sufficiently insisted upon by writers, that *empyema* does not give rise to hæmoptysis. In sixteen well-marked cases which I have had under my care within the last two years, for periods of three months and upwards, no single example of hæmoptysis occurred. But more than this, empyema, established in a phthisical person, seems to be *preventive of hæmoptysis*. In seven cases of combined tuberculous excavation and empyema, carefully watched, and proved as to the diagnosis, either by dissection or by indubitable signs, no spitting of blood had ever occurred. All these seven persons were males. The pressure exercised on the lung by the contents of the pleura might appear to explain the fact plausibly enough, especially as the excavations were on the same side as the empyema in six of the seven cases; and I have recently seen a curious exemplification of the apparent influence of excessively acute pleuritic effusion in arresting obstinate hæmoptysis. But, on the other hand, in one case, excavations existed in both lungs.

"(*d.*) *Simple chronic consolidation of the lung* has not, in my experience, been attended with hæmoptysis to any amount.

"(*e.*) *Acute pneumonia*, accompanied with discharge of pure blood, is almost positively connected with tuberculous disease.

"(*f.*) *Gangrene* of the lung is rarely attended with hæmoptysis in the adult; in infancy (when tuberculous hæmoptysis is very rare) it is rather common.

"(*g.*) I have never known *ulceration of the larynx* productive of discharge

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"* Pathologically, these people were *latently* tuberculous persons, with superadded bronchitis; but practically, they could only be regarded as bronchitic."

of blood to any extent; streaks are not uncommon. But ulceration of the larynx (proceeding from within outwards) seems not to occur as a primary affection; at least I have never seen it except in phthisis (which may be completely latent in regard of the lung), cancer, and syphilis.

"(*h.*) Hæmoptysis arising from *disease of the heart* can with difficulty be confounded, even in itself, with the severer forms of phthisical hæmorrhage; while the physical signs of the cardiac disease will point to its true source in such cases of the slighter form. I have never once seen cardiac disease, of such kind as to cause hæmoptysis, coexistent with *phthisis,* using the term in its practical sense; but in a fair number of instances I have seen advanced cardiac disease in persons whose lungs contained *crude tubercles and grey granulations.* It may be, therefore, that the conditions of the system existing in heart-disease are unfavourable to the development of tubercle; but the infrequency with which the two kinds of disease are found together doubtless depends, in the main, on the difference in the periods of life at which each is especially prone to occur.

"(*i.*) *Aortic aneurism,* opening into the trachea, may (without proving immediately fatal) give rise to hæmorrhage, indistinguishable by its own characters from profuse pulmonary hæmorrhage. The history of the case, the physical signs, the age of the individual, &c., commonly establish the diagnosis; but when the aneurism is small, and so placed as to elude percussion, and pressure-signs, both concentric and eccentric, are absent, the difficulty of *proving* the existence of aneurism may be insurmountable; the existence of the disease may be divined, but not demonstrated. It is to be remembered that the absence of notable signs of tuberculisation does not justify the inference that the hæmoptysis is not phthisical, seeing that a tremendous pulmonary hæmorrhage may occur when slight consolidation exists at one apex only, and that such consolidation might be supposed to depend on the pressure of an aneurism.

"(*k.*) It is matter of common belief that in women who menstruate imperfectly and irregularly, the expectoration of a small quantity of blood is insignificant. I think this *perhaps* true where streaks only are concerned; but in every instance I have observed (*except one*), where such succedaneous hæmoptysis reached an ounce or upwards, there has been either evidence, or ground for suspicion, of tuberculisation. Similarly I have seen two cases of individuals presumedly in a state of perfect health, who, in a violent fit of passion brought up a certain quantity of blood from the lungs: *both had latent tubercles.*

"The tendency of my experience, then, is clearly to show the vast frequency with which hæmoptysis is in some manner or other an attendant on tuberculous disease. The fact, that individuals are occasionally met with who, after having had more or less profuse hæmoptysis, live on to a good age without exhibiting phthisical symptoms, does not invalidate this result; it simply shows that tuberculisation tending to hæmoptysis may, as well as that not so tending, undergo spontaneous suspension."

The *diagnosis* of hæmoptysis in general may be considered in the present place; while we reserve for future consideration any special points connected with its distinction, in particular diseases of the lungs and heart.

Hæmorrhage from the mouth and fauces can be distinguished by careful inspection of these parts from hæmoptysis. Epistaxis, under ordinary circumstances, cannot be confounded with hæmoptysis; but sometimes blood, instead of coming forwards from the nares, trickles backwards, and may be from time to time coughed up. But here, again, close examination of the nares anteriorly, and of the pharynx, will disclose the source of the hæmorrhage.

Hæmoptysis is distinguished from hæmatemesis by the following characters. Hæmoptysis is preceded by slight dyspnœa, anomalous sensations about the chest, tightness, weight behind the sternum, or at some other spot, to which the patient will sometimes confidently point as the seat of mischief; hæmatemesis by weight and uneasiness at the epigastrium, sometimes by nausea. When the lung supplies the blood, the pulse is oftener excited, full, bounding, sometimes *bisferiens*, than when the stomach is its source. A salt taste in the mouth, with tickling and guggling sensations in the throat, often precedes actual hæmoptysis: whereas I certainly have never known this complained of in hæmatemesis. Blood is ejected in severe hæmoptysis with efforts indistinguishable by patients from those of true vomiting; but previously to such "vomiting of blood," mouthfuls have generally been coughed up. Hæmatemesis is attended and followed by tenderness at the epigastrium; hæmoptysis by none of this. No matter what quantity of blood be brought up from the lungs at once, small quantities continue to be expectorated for a time; when the stomach is at fault, on the contrary, full discharge occurs suddenly, and there is, generally speaking, an end of the matter,—certainly no bloody or stained sputa follow. In hæmoptysis the blood is florid and frothy; in hæmatemesis dark and non-aërated: at least this is the common case. But when large masses of blood are discharged from the lungs, they may be totally frothless; and where hæmorrhage occurs rapidly from an artery into the stomach, the blood is vomited at once, and of perfectly arterial hue: no time is allowed for discoloration by the gastric fluids. Discharge of blood by stool is the rule in hæmatemesis; the exception in hæmoptysis: in the latter case, it comes of blood accidentally swallowed, and is never, so far as I have known, abundant. In hæmoptysis liquid rhonchus may almost invariably be found in some part of one or both lungs; nothing of the sort exists in hæmatemesis; in the former, the pulse is proportionally less quickened than the respiration; still this perversion may occur in hæmatemesis also. Lastly, the diagnosis should never be fixed on without inquiring into the history of the patient, and making a cautious examination of the chest. Should the evidence of chronic changes at the apices exist, a doubtful opinion in favour of pulmonary origin would at once be strengthened; but the absence of such changes would not exclude the possibility of hæmoptysis; for, as will hereafter fully appear, such discharge of blood may occur before any notable physical changes have occurred in the lungs. The state of the chylopoietic viscera should be examined physically in aid of the diagnosis of hæmatemesis.

The *treatment* of hæmoptysis, during its actual existence, aims (*a.*) at removing the conditions causing it, or (*b.*) at stopping it in spite of those conditions. (*a.*) Now if there be evidence of congestion of the lung of an active kind, with febrile excitement and strong cardiac action, that congestion should be treated by bleeding from the arm, to an amount measured by the urgency of the symptoms, and the constitution of the individual. Slow local bleeding from the chest by leeches I believe to be highly objectionable; the rapid abstraction of blood by cupping, however, if the patient be enfeebled by previous disease, is preferable to venesection. The head should be kept high during the bleeding; and, indeed, throughout the progress of the case; a semi-faint state tends in itself to control hæmorrhage. Leeches to the anus, or to the feet, followed by the hot pediluvium, sometimes very manifestly control hæmoptysis, where there are evidences of abdominal congestion. Nauseating doses of tartarised antimony, or of ipecacuanha, are, by some had recourse to from the first; but the practice is one of which I have little experience. Tartarised antimony, it is affirmed on high clinical authority, has actually caused death under these circumstances: however, it does not necessarily increase bleeding, even though it causes vomiting,—this I have *seen* in the practice of others. The bowels should be freely opened with cooling saline purgatives, and watery evacuations, if possible, be kept up for a day or two.

Ligature of the limbs, so as to prevent the free return of blood through the veins, has proved a useful adjuvant occasionally. Raising the arms over the head unquestionably stops epistaxis sometimes; I know not what effect the position may have in hæmoptysis. Free circulation of cool air, light bed-clothes, a hard bed, quietude, and silence are essential aids. Large pieces of Wenham Lake ice should be allowed to dissolve in the mouth; and the *cautious* application of ice in bags to the spine or over the heart I have repeatedly seen (hence I do not value the speculative objections to the practice) almost instantaneously arrest the flow of blood. Heat may at the same time be applied to the extremities.

Among remedies, controlling the action of the heart, digitalis, aconite, and prussic acid claim attention; if the heart be irritable, and the hæmoptysis moderate, the first-mentioned medicine is valuable. Refrigerants, such as nitrate of potass, sulphuric acid, &c., may be employed as adjuvants.

(*b.*) The medicines belonging to the astringent class, in which I place most confidence, are the acetate of lead, given in doses of two grains with dilute acetic acid and laudanum, every half-hour, hour, or two hours, according to the urgency of the case; gallic acid (grains, three to six, as a dose); alum; ergot of rye (not so valuable, however, as in epistaxis) and matico. If there be excessive anæmia, the tincture of the sesqui-chloride of iron, or sulphate of iron with gallic acid (making a gallate of iron) may be given from the first. In various other astringents, krameria, logwood, kino, catechu, sulphates of zinc and copper, little trust is to be reposed; nor, useful as it is in some hæmorrhages, have I ever seen turpentine distinctly beneficial in hæmoptysis. Drachm doses of kitchen salt, either in powder or dissolved in water, appear sometimes (I have seen the fact in three instances) to arrest hæmoptysis very rapidly,—and this, whether they produce emesis or not. Five or six drachms may be given at intervals,—and, as the agent is always at hand, it may at once be used, while other means are in preparation.

The treatment of hæmorrhagic reaction and of sinking is the same in the case of hæmoptysis as of all hæmorrhages.

Are there any means of preventing the tendency to frequent hæmoptysis in the course of tuberculous disease? I believe not. Cheyne, of Dublin, it is true, had great faith in the prophylactic virtues of small and repeated bleedings; but I confess that the case cited from his practice by Sir James Clark hardly makes me a convert. Hæmorrhage was clearly not prevented in this instance (though a *weekly* venesection was performed for a year), for the bleedings recurred again and again; and blood-letting seems eventually to have failed even to control the seizures, when actually present. I fully agree with Sir James Clark, that the remedy was relied on too exclusively; nor can I believe that this patient's case was the type of a class, for, instead of becoming spanæmic and emaciated, as the mass of men would under such treatment, he appears to have grown into flesh and regained strength.

ALTERATIONS OF SECRETION.

Œdema of the Lung.

Œdema of the lung, or serous infiltration of its parenchyma, is in the immense majority of cases a *secondary* state, occurring either as a part of general dropsy, as a dependence on disease of the heart, as a sequence of congestive conditions of the lung (as after continued fever), of acute and chronic bronchitis, or of pneumonia. Laennec taught, however, that it may occur as a *primary* and *idiopathic* condition, and that the suffocative orthopnæa which sometimes cuts off children after measles, arises from such œdema. Even as a secondary condition it is rare—at least, it is rarely demonstrable *post mortem.*

Disturbance of respiration from a slight to an intense degree, slight cough, watery, or sometimes rather tenacious, expectoration, sensation of weight and heaviness within the chest, constitute its symptoms—a combination anything but distinctive; neither are the physical signs conclusive. Inspection discloses nothing sufficiently marked to be trusted to; the vocal fremitus may be slightly intensified; the percussion-sound is duller than natural; the parietal resistance increased; the vocal resonance varies in character; the respiration is weak, and harsh, or even blowing, and mingled with liquid subcrepitant rhonchus: the last-mentioned, when well marked, is the most distinctive, sign.

In congestion of the lung the subcrepitant rhonchus is drier than in œdema, the expectoration more viscid, and there are no dropsical symptoms. Hydrothorax is unattended with rhonchus, and the dulness, caused by the pleural fluid, changes its seat with the posture of the patient. Pleural pseudo-rhonchus, unless care be taken, may be confounded with the subcrepitant rhonchus of œdema (vid. p. 120); the rhonchus of capillary bronchitis is rather to be distinguished, it must be confessed, by coexistent evidence of bronchial inflammation, than by its own characters.

Œdema, occurring after pneumonia, furnishes an indication for the use of gentle tonics. If it form a part of general dropsy, it is mainly to be relieved by means calculated to lessen the latter. But dry-cupping and a succession of flying blisters to the chest, sometimes exercise a distinctly beneficial local effect.

HYDROTHORAX.

Hydrothorax, or dropsy of the pleura (serous fluid without inflammation-products), occurs actively, passively, or mechanically. Of the former kind is the true hydrothorax, occasionally putting the close to existence in cancer of the mamma, and also occurring in *certain* cases of Bright's disease: on the whole, this variety is very rare. In the great majority of cases, hydrothorax is passive or mechanical, occurs as a part of general dropsy, or is produced by obstructed circulation through the lungs and heart,—especially the right side and tricuspid orifice.

In hydrothorax the pain and "stitch" of pleurisy are wanting, and there is no tenderness under pressure. There is less cough, and may actually be none. But the mechanical effects of hydrothorax are commonly more serious than those of pleuritic effusion, for the simple reasons, that hydrothorax is generally double, and sequential to more or less serious organic disease, already disturbing the respiration and circulation,—pleurisy, on the contrary, generally single and primary. Hence the dyspnœa may be excessive, with constant orthopnæa, and extreme lividity of the face, anxious countenance, clammy perspirations, and coolness of the expired air. If the patient can lie down, he does so on the back, with slight inclination now to one side, now to the other.

The physical signs *agree* with those of pleurisy in some, *disagree* in other points. Dulness under percussion, moveable in area, with the changed posture of the patient; distant, weak, or suppressed respiration; occasional ægophony; total loss of vocal fremitus, and occasionally peripheric fluctuation, are common to the two affections. But in hydrothorax there is no friction-sound or fremitus, and both sides are commonly affected, while in pleurisy one only generally suffers. Dilatation of the side and flattening of the intercostal spaces exist in both diseases, but are carried habitually to a higher point in the inflammation than in the dropsy. The heart and mediastinum are but little displaced sidewards in hydrothorax, because the disease is generally double; the diaphragm may be very considerably depressed.

If the dropsy be of the irritative kind, moderate cupping (unless some contra-indications exist) is beneficial; under all circumstances, dry cupping is advisable. If there be no marked œdema of the walls, the chest should be blistered. Painting with caustic iodine, or ioduretted frictions, sometimes obviously promote absorption. The internal remedies are diuretics and purgatives.

Inasmuch as hydrothorax is commonly a *local* manifestation of a *general* disease, little, as a rule, is to be expected from paracentesis,—at least in the way of permanent cure. But great temporary relief, and even prolongation of life, may be secured in urgent cases by the operation; and, where asphyxia is threatened by double hydrothorax, it appears to me that puncture should at once be had recourse to.

PNEUMOTHORAX.

I.—Decomposition of solid or fluid materials within the pleura, and the secretion of gas by that membrane, are alleged causes of simple non-perforative pneumothorax. As already mentioned, I believe cases of pneumonia occur in which a local pneumothorax appears referrible to a process of secretion; of the other kinds of simple pneumothorax, I know nothing clinically. In the immense majority of cases, pneumothorax comes of injury to the walls of the chest, or of perforating disease of the lung; and probably in 90 per cent. of the latter class of cases the perforation is tuberculous.*

II. Practically, then, the symptoms of invasion of pneumothorax (excluding traumatic cases) are equivalent to those of perforation of the lung, or, more correctly speaking, of the pulmonary pleura. And these are sudden sharp pain in the side, often of agonising, overwhelming intensity, coupled occasionally with a sensation of something having given way internally, and of fluid escaping into the chest, and almost invariably most intense dyspnœa. If these three symptoms suddenly and unmistakeably appear in a phthisical person, the diagnosis of perforation would rarely be at fault; but I have known perforation occur, as proved by physical signs and inspection after death, without any one of the three announcing its occurrence. The physical signs are the only *unfailing* evidence. After a time, the sensation of dyspnœa may wear off; I have known a man's respirations 52 in the minute without his feeling any of the sufferings of difficult breathing.†

Air effused into the pleura acts as an irritant on the pleural surface; fluid is produced (often within twenty-four hours), and the compound state of hydro-pneumothorax is established. The patient lies in various postures, but most commonly and steadily on the back, inclining to the sound side, with the head more or less raised: orthopnœa also occasionally exists. The pulse is excessively frequent; but the respiration relatively more so: I have known the ratio perverted into one of 2·3 to 1. The countenance is pinched, anxious, and imploring; the lips, cheeks, and face generally more or less livid; the sleep consists of fitful dozes at rare intervals; the skin is moist, sometimes bathed in cool clammy perspiration. The voice habitually loses strength greatly, and almost complete aphonia has sometimes been observed. Œdema of the affected side of the thorax is, at the least, very rare, before pneumothorax has been accompanied, and this for some time, with pleuritic effusion.

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* Of 147 cases of pneumothorax collected by M. Saussier, 81 only were phthisical. But tuberculous perforation is an every-day affair, which passes unnoticed; perforations from gangrene, vesicular emphysema, hydatids, pulmonary apoplexy, abscess, &c., are greedily caught hold of, and recorded. The number of the latter *published*, consequently, gradually swells out of proportion with their real frequency.

† Plimpton, Clin. Lect., loc. cit., p. 575.

The perforation may either undergo closure by lymph, or remain pervious. I have seen two cases of the former kind, in which all signs of air and fluid in the pleura had disappeared in the course of two months after the perforation. Even when the opening remains pervious, the compound disease is not necessarily fatal. Laennec refers to a case where the signs of fistulous hydro-pneumothorax continued at the end of six years. M. Louis, and the French school, generally, have taken a too unreservedly gloomy view of the prognosis of phthisical perforation; for cases have now been collected in some numbers in this country positively proving that not only may life be prolonged, but excellent health enjoyed, while succussion-sound is well audible in the side: still such cases are completely exceptional.

III. The physical signs of *pneumothorax* are very significant. The chest-motions suffer more or less extensively in freedom; they may be absolutely null at the lower part of the affected side: when there is any play, the intercostal spaces deepen during inspiration greatly. The vocal fremitus is weakened or annulled; the width of the side increased to the eye and to measure; the interspaces widened, and even bulged outwards, inferiorly, may be natural superiorly. The percussion-sound, increased in clearness, acquires tympanitic quality, retaining this until the accumulation of air becomes so great as to check the vibration of the walls under the blow. Local pneumothorax, at least in front of the trachea and large bronchi, may give an amphoric note. If the quantity of air be moderate, the respiration is of distant, weak type,—if considerable, absolutely suppressed. The conditions of vocal resonance vary: there may be mere nullity of sound; in some cases, the resonance is loud and diffused; and possibly it may be sometimes accompanied with metallic echo. The heart's sounds are, as a rule, obscurely transmitted through the air in the chest; and the mediastina, heart, and diaphragm displaced. The clearness of percussion may extend considerably beyond the middle line.

In cases of simple hydro-pneumothorax the signs are a combination of those of pleuritic effusion and of pneumothorax,—the former at the lower, the latter at the upper, part of the side.

When hydro-pneumothorax coexists (as is the rule) with perforation of the lung, fluctuation may be felt both by patient and observer, when the chest is abruptly shaken; peripheric fluctuation exists to its maximum amount; and Hippocratic succussion-sound is readily produced: these three signs may exist, although closure of the perforation has taken place. The dull sound of fluid, and the tympanitic resonance of air, are found, in the ordinary posture of patients, the former inferiorly, the latter superiorly: but the exact sites of both may be variously changed (unless adhesion, which is rare, interfere) by altering that posture; the respiration is amphoric, with or without metallic echo or tinkling; and the cough and vocal resonance are similarly echoed. The phenomena of displacement of organs are carried to the highest possible point. The heart's sounds are commonly weakened in their passage across the distended pleura; but they are sometimes echoed within it. A peculiar inspiratory sibilus is sometimes heard all over the side, and probably depends on escape of air through the chink in the lung.

IV.—The *treatment* of perforative tuberculous pneumothorax is palliative. If the patient be seen immediately, or shortly after the pleura has given way, while his agony exists in all the intenseness of novelty, bleeding suggests itself as a means of relief. The quantity of oxygenating surface has been suddenly reduced, and the sudden disparity between that surface and the mass of the blood might, or would, I think, be somewhat lessened by diminishing the quantity of the latter. Whether this be the explanation or not, venesection does give very notable relief, and renders subsequent inflammation of the pleura less violent. It should then be had recourse to, where the patient's strength has not been materially impaired by the previous disease. In doubtful cases, moderate cupping of the side may be substituted,—or if even this be feared, dry cupping of the chest generally. It is singular what relief, both of pain and dyspnœa, is sometimes afforded by the latter process. Repeated flying blisters to the side are also most valuable agents. The bowels must be kept moderately open, perspirable action of the skin promoted, the strength maintained by nutritious animal jellies and broths, or meat, if the patient's digestive powers be not enfeebled, and inflammatory symptoms controlled as they arise.

Various anti-spasmodics are useful in mitigating the dyspnœa, —lobelia inflata, cannabis indica, belladonna, stramonium, aconite, and, above all, opium. I have seen musk, in five grain doses, afford great relief.

In tuberculous pneumothorax, paracentesis can only be regarded as palliative, and, what is worse, temporarily palliative. Still, as the operation does not obviously place the patient in any way in a worse position than he had been before, and as it often gives new existence for a while, there can be no objection to its employment (and to its repetition), when physical signs show that the mediastinum and the non-affected lung are seriously encroached upon.

It is difficult to lay down a rule for other varieties of perforative pneumothorax; recorded cases are deficient in detail for our guidance, and to me it appears that where recovery has ensued, it would have occurred without the operation. If pneumothorax were suddenly produced in a fit of violent coughing, as in hooping-cough, and the patient had previously exhibited no evidence of organic disease in the lung, I should hold it advisable (especially if the symptoms were urgent) to puncture the thorax.

DISEASES OF NUTRITION.

EMPHYSEMA.

I. THE disease, inconveniently termed vesicular emphysema by Laennec (and which it might be better, perhaps, to call *rarefaction of the lung*), is essentially characterised by enlargement of the air-cells, obliteration of their vessels, and atrophy of their walls; occasionally oil (as first pointed out by Mr. Rainey); is discoverable in the walls of the vesicles, but it is not constant,—and when present, its relationship (of cause or of effect) to the existing atrophy is uncertain. A state of hypertrophy of the inter-vesicular structure occurs in very rare instances.

II. The sole symptom of atrophous emphysema *per se* is dyspnœa. Often commencing in early youth, or even infancy, at first slight in amount, and only felt on some unusual exertion, such as running up-stairs, &c., when once developed, dyspnœa is permanent, but subject to great variations in intensity. It is true, many persons labouring under emphysema will affirm that their dyspnœa is only occasional,—that habitually their respiration is perfectly natural. But I have never known an instance of this kind where the patient was not the victim of a delusion; the truth is, that a moderate amount of dyspnœa had become to him second nature, a state of comfort and health,—and excessive difficulty of breathing alone gave him annoyance. The dyspnœa is increased, from time to time, either through spasm, through abdominal infarction, or through intercurrent bronchitis; the former two causes produce sudden paroxysmal attacks, requiring the patient to rush to the open window for air, or pass the night in the sitting posture out of bed; the latter is less violent, but more protracted, in its action. The amount of persistent dyspnœa is generally proportional to the duration of the disease. The paroxysm is occasionally accompanied with a fit of palpitation, but not unless some cardiac disease co-exist. Cough preceding, commencing with, or following dyspnœa, exists in almost every case; the habitual sputa are frothy, liquid, and mucous or watery. It is matter of doubt whether the state of the lung itself, independently of that of the bronchi and pleura, may give rise to pain; my own observation leads me to doubt it. The facies of emphysematous patients is peculiar: of dusky colour, and anxious melancholy expression, it is full, out of proportion with the chest and body generally,—a probable result of thickening of the cellular membrane and muscles of the face, as suggested by Dr. Stokes,—the former from repeated venous obstruction, the latter from respiratory effort. The nostrils are thick, and very often the lower lip full, and venously turgid: I have, however, not seen the latter state without heart-disease. The muscles of the neck enlarge, and its cellular tissue disappears. The patient's gait is stooping; Dr. Stokes has known the acromial, interscapular and lower scapular regions almost horizontal. The strength is inversely as the dyspnœa; in aggravated cases, bodily exertion becomes an impossibility. The flesh of the body generally and slowly fails,—each attack of bronchitis diminishing the weight *pro tempore*, or permanently. The pulse is not accelerated,—far from this, it ranges below the average of health in a considerable number of cases, except when intercurrent bronchitis is present. The respiration also (with the same qualification) is less frequent than in health; in fact, the act is so laboured a one, that it cannot be often repeated in the minute. The pulse often strikes the observer by its weakness, as compared with the amount of cardiac impulse. This want of accordance comes of the frequency with which the right heart undergoes enlargement (while the left does not suffer), as a consequence of the pulmonary disease. No conceivable amount of emphysema of both lungs will necessarily entail the smallest amount of dropsy,—even œdema of the ankles. If dropsy occur, there is something else (generally tricuspid regurgitation) to account for it. The bowels are habitually constipated; the urine aqueous.

Emphysema tends, in the course of years, to produce dilated hypertrophy of the right heart, Bright's disease, and habitual inclination to venous congestion within the head. It is rather protective than otherwise against tubercle, has but slight influence in producing dilatation of the bronchi or pleurisy (Louis), rarely leads to interlobular emphysema, and still more rarely, by rupture of a sub-pleural sacculus, to pneumothorax.

III. The physical signs of emphysema are numerous and positive. Inspection discovers bulging of the infra-clavicular, mammary, and central sternal regions, or of the anterior surface generally. General expansion of the chest occurs very rarely; M. Louis observed it only once in ninety-six cases: when it exists, it gives the chest a globular form.

The state of the interspaces in the bulged portions of surface has been matter of dispute. Dr. Stokes has taught that in emphysema, "even after great dilatation of the chest has occurred, we see the intercostal spaces, so far from being obliterated, *deeply marked;*" and that the single malady in which this obliteration really occurs is pleurisy in its advanced stages. The conditions directly conducive to its production are *paralysis* of the intercostal muscles and excentric pressure, one being as essential as the other; this paralysis is presumed to be the result of inflammation extending to the muscular tissue. For the same reason, the intercostal spaces will not be obliterated in cases of simple hydrothorax, nor in *all* instances of pleuritic effusion; because muscular inflammation and paralysis do not exist at all in the former, and are not *necessarily* present in the latter. The question here started is strictly one of observation; and it must be confessed that the experience of physicians generally does not accord with that of Dr. Stokes in respect of the bulging of emphysema. MM. Louis and Woillez (not to mention others who have paid less special attention to the form of the chest in emphysema) are wholly opposed to Dr. Stokes on this point. Both maintain that the intercostal hollows are in this affection either effaced or manifestly less marked than in the natural state; and even point out this implication of the muscular plane of these spaces as one of the distinctive marks of emphysematous prominence. In point of fact (as mentioned several years ago), I believe that in emphysema the conditions in respect of bulging are of three distinct kinds. *First*, there may be no expansion at all, general or local; *secondly*, there may be bulging of the surface generally, with a natural state of the intercostal spaces; and *thirdly*, there may be bulging with distinct obliteration of the intercostal hollows. The key to these apparent contradictions lies mainly in the anatomy of the disease—in its anatomical varieties, which have in this point of view escaped the consideration of the observers referred to. In the *first case*, I have found the disease, which, in respect of symptoms, may have been very intensely marked, of the *atrophous* kind, with but *slight* distension of the lung; here the physical cause of expansion was altogether wanting. The *second variety* of prominence I have never observed in the infra-clavicular region (when alteration of shape was limited to that region, one of the special seats of such change in emphysema), but have met with it elsewhere in certain cases of almost globular expansion of the thorax in emaciated emphysematous subjects. But in these instances—and doubtless they exist more frequently than they are discovered or suspected—the local prominence (when characterised in the manner now referred to) was in all probability, especially when occurring at the back, a natural conformation, and wholly *independent of the emphysema. Thirdly*, when bulging has existed in regions where observation proves it to appertain specially to emphysema,—*e. g.* the infra-clavicular,—I have found the intercostal spaces distinctly prominent, and the disease, if opportunity for *post-mortem* inquiry presented itself, either of the *hypertrophous* kind, or of the atrophous variety, with great distension of the lung and formation of sub-pleural sacculi of air.* The inspiratory motion of expansion is greatly diminished; the lower part of the sternum and adjoining cartilages, in aggravated cases, even sinking inwards during inspiration,—nay, the base of the chest generally may slightly retract. On the other hand, the elevation-motions are exaggerated,—the whole chest is raised, with the shoulders, as one piece. The duration of the expiratory movement considerably exceeds that of the inspiratory; both acts are laborious,—the expiratory, because the elasticity of the lung is destroyed by the disease,—the inspiratory, because the previous expiration has failed to empty the lungs to the normal amount, and the chest is with difficulty further dilated. The faculty for prolongation of the expiratory act, is probably derived from hypertrophy of the muscular coat of the bronchial tubes;—their expressing power is permanently taxed to the utmost.

The condition of vocal fremitus varies; it may exceed the average of health. The semi-circular measurement of one side, or of the whole chest, is increased; in right-handed emphysematous persons, as first noticed by M. Woillez, the excess of right width over the left ranges lower than natural,—showing by inference the greater frequency of emphysema of the left side. The disease may, however, be highly marked without any such increase. The percussion-sound is greatly clearer than natural, of exaggerated pulmonary, more or less tympanitic, quality, while the resilience of the chest-walls is augmented. The morbid clearness extends beyond the middle line, if one lung only be affected. The natural resonance at the sternal notch, where no lung exists in health, is exchanged for emphysematous clearness; this fact, which depends on the junction of the edges of the lungs taking place higher than natural, in consequence of their distension, is very easily ascertained, both in the living and dead. Forced expiration has little or no effect in diminishing the amount, or contracting the area, of the morbidly clear resonance.

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* The difference of opinion under consideration appears to be very readily explicable in the manner now proposed. What I have stated I believe to be in strict conformity with observation; whereas I am not aware that Dr. Stokes's theory, of inflammation of the intercostal muscles being a necessary condition of their excentric displacement, rests upon any observed cases, submitted to close anatomical examination. It is not easy to understand, in his theory, why the intercostal muscles should resist excentric pressure more powerfully than the ribs; and this, be it noted, equally in subjects of every degree of muscular weakness or vigour. It appears to me that there will always be more or less hollow in the intercostal spaces, as has been shown by M. Woillez, so long as the elasticity or concentric force of the lung is not destroyed; that as soon as this change has taken place, as, for example, from the progress of emphysema, pressure sets in and influences the position of the intercostal muscles at least as readily as that of the ribs.

The respiration in emphysema belongs to one or two main types; it is weak, or of raised pitch, and altered quality. When of weak type, the failure may amount to actual suppression in some points; while in others, exaggerated respiration of harsh quality exists. Of the occasional existence of such exaggeration in lung-substance, on the confines of circumscribed emphysema, I have satisfied myself by a considerable number of observations; but, on the whole, it is rare. Respiration of raised pitch, loud, harsh, and uneven, exists, in many cases, immediately over emphysema carried anatomically to the very highest point; the quality is sharply sibilant, and in part it may be constituted by a sibilant rhonchus in the very finest tubes. The inspiratory and expiratory sounds are separated by a distinct pause (*divided* respiration); the former is comparatively short, the latter greatly prolonged: the ratio of inspiratory to expiratory sound may be changed from 3 : 1 to 1 : 4; in other words, the latter sound made *twelve* times longer in proportion to the former than in health. In such cases of enormous prolongation of expiratory sound, this is rather a fine sibilant rhonchus than true respiratory murmur. Rhonchi, bronchial, and dry or moist, announce co-existing bronchitis; subcrepitant rhonchus may also be heard at one or, more commonly, at both bases. Dry grazing friction-sound may be produced by sub-pleural sacculi. The vocal resonance varies; it may be null or bronchophonic. The heart's sounds are feebly transmitted through the emphysematous texture.

Signs of importance are derived from the heart. If one lung only be affected, the heart is pushed slightly to the opposite side; if both are implicated, downwards, and to the right somewhat. The præcordial region, filled with distended lung, is bulged forwards; it sounds clear under percussion; no cardiac impulse may be felt within its area,—that impulse being transferred to the epigastrium. Enlargement of the right side of the heart, however, plays its part in thus altering the seat of impulse. The jugular veins may be swollen, though the heart is not obviously diseased; but I have never known them pulsatile, without co-existent affection of the heart. Emphysema is sometimes accompanied with visible arterial pulse: but I have not seen this, unless the patient were aged, or the subject of aortic regurgitation.

Are there any special signs of interlobular emphysema? I know of none. It is quite possible, as Laennec originally taught, that, if the surfaces of the interlobular spaces become prominent through distension, grazing, dry, friction-sound may be heard; but there will be nothing distinguishing the sound thus produced from that caused by the sub-pleural sacculi (or so-called vesicles) of advanced vesicular emphysema. The same observations apply to Laennec's "dry, crepitant rhonchus, with large bubbles."

IV. The only affection with which emphysema can be physically confounded, is pneumothorax. The distinction is not difficult. In emphysema the percussion is less clear, less truly tympanitic than in pneumothorax; the side is less dilated; there is more respiration (and, possibly, loud, sibilant, respiration, as we have seen), and such respiration as exists is superficial, instead of being deep-seated and distant, as in pneumothorax. Besides, pneumothorax affects only one side (double pneumothorax must be almost instantly fatal); whereas if emphysema exist to such a degree as to simulate in regard of percussion-sound the presence of air in the pleura, both lungs are seen to be very seriously affected. Lastly, the symptoms of pneumothorax come on suddenly, and are greatly more urgent than those of emphysema.

V. The anatomy of emphysema prepares us to fear that its radical cure is impossible; we have no means of causing reproduction of destroyed lung. Experience, unfortunately, goes farther than this, and shows that *permanent* palliation even is most difficult of accomplishment: *temporary* relief is, on the contrary, most readily effected: the fair aim of art is, then, to ascertain, how a state of brief ease may be indefinitely prolonged.

The proofs that great amelioration is actually produced by treatment, are furnished not only by favourable change in the symptoms and in the patient's feelings, but by changes in the physical signs: the morbid clearness of percussion may be lessened, the quantum of respiration increased, and the distension of the lung reduced; a fact positively demonstrable by increase in the area of the heart's superficial dulness.* But what organic change in the lung-substance do these alterations in physical signs demonstrate? Not, assuredly, that atrophy is gone, but that *distension* is reduced. And the reduction of distension is effected by the removal of bronchitis and the relaxation of spasm.

In nine cases out of ten, when a sufferer from emphysema applies for relief, sub-acute; rarely very acute, bronchitis is present. Local bleeding, blistering, and small doses of tartar emetic are the essential agents for its removal. The other remedies, spoken of under the head of bronchitis, are not to be forgotten, however, when the first vigour of the intercurrent disease is controlled. Where emphysema exists to any notable amount, depletory measures must always be cautiously employed.

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* *Vide* case of Hope, Clin. Lect., loc. cit., April, 1849.

For the habitual chronic bronchitic state, I know of no combination superior to the æthereal tincture of lobelia inflata, with ipecacuanha, in ammoniacum mixture. Fits of dyspnœa are to be relieved by extract of cannabis indica in half-grain to grain doses, or by belladonna and stramonium, either in the form of pill or smoked in cigarettes. Stramonium alone may be smoked in an ordinary pipe, and may be more safely entrusted to patients than belladonna: it may be smoked until the head begins to ache slightly. Opium, however, must be had recourse to in very severe attacks, and may be pushed until sensible, but slight, narcotic effects are produced.

Transmission of a gentle galvanic current from the nucha to the epigastrium, will sometimes avert, almost invariably mitigate and postpone, the paroxysm of dyspnœa.* An emetic (sulphate of zinc) will do so also; especially when the attack is connected with a loaded state of stomach. Cajuput oil and other carminatives, with soda, put a term to that caused by flatulent distension.

Strychnia has been recommended for the purpose of improving the tone of the non-striated fibre of the bronchial tubes. I have tried it in a small number of cases, both endermically and by the mouth, in sufficient doses to produce obvious effects on the voluntary muscles, without in the slightest degree modifying the symptoms of the emphysema.

Regularity of habits, and moderation in indulgence at table, are essential to the well-being of emphysematous sufferers; and it is obvious, from their forced obedience in regard of these points, that their lives are often (a questionable boon, it may be, as they themselves have more than once said to me) prolonged beyond the average term of existence.

Change of air is singularly beneficial, or singularly detrimental, according to the nature of the change; but no amount of experience will enable the physician to predict positively what manner of air will best suit any individual case. In order to avoid repetition, I defer the illustrations of this point to the head of "Spasmodic Asthma."

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* *E. g.* Case of M. Henley, Males, U. C. H., April, 1851.

HYPERTROPHY OF THE LUNG.

Hypertrophy of the lung, unless when accidentally ensuing upon emphysema, cannot be regarded in the light of a disease. It arises as the result of inaction of the fellow-organ, as when this has been compressed and rendered permanently useless by empyema. The side may become considerably dilated, the percussion-sound clear, the vocal fremitus marked, the vocal resonance strong, and the respiratory sounds exaggerated and harsh, with an undue amount of expiration. Emphysematous distension of the air-cells sometimes advances *pari passu* with hypertrophy.

PULMONARY TUBERCLE.

Tubercle in the lung constitutes the anatomical character of phthisis, or consumption,—a disease which, in the vast majority of cases, runs a chronic, but occasionally an acute course.

CHRONIC PHTHISIS.

The anatomical changes occurring in the lung in chronic phthisis are referrible to three main stages; and these stages correspond habitually to certain varieties in the symptoms, and always to modifications in the physical signs. There are, then, clinical as well as anatomical reasons for recognising three stages in the disease,—those of deposition and induration, of softening, and of excavation.

I. The *first stage*,—that of accumulation of groups of grey granulations or crude tubercles in variable quantity, and with or without intervening simple induration of tissue,—has the following physical signs.

The infra- and supra-clavicular regions on the affected side (granting that one side only is affected) are either unaltered in form or they are slightly flattened. The former is the more common of the two cases: probably, the diminished bulk of the apex, produced by deposition of tubercle and closure of *some* air-cells, is at first counterbalanced by the distension of *others*. It falls within the limits of the probable, indeed, that cases may now and then be met with, in which such emphysematous distension shall so predominate as to produce slight bulging over tuberculised lung. Many years ago, Dr. Chambers, without offering any explanation of the circumstance, mentioned to me that he had observed an increase in the antero-posterior diameter of the apex at the very outset of the disease. I have carefully watched for examples of the fact, but have never met with one, unless where the sound was clear under percussion, and the existence of local emphysema, therefore, strongly probable. I hold it, as matter of experience, to be impossible that tubercle, accumulated to such an amount as to impair in the least degree the clearness of percussion-sound, shall be the cause of bulging. The precise amount of tuberculisation, which will produce flattening, varies with many accidental circumstances, such as the relationship of the tubercles to the minuter bronchial tubes (if many of these be obstructed, local collapse of lung-substance will ensue), and the presence or absence of plastic contractile exudation within the parenchyma or on the pleural surface. Atrophy of the tissue of the lung can scarcely occur at the very earliest stage of bronchial obstruction. Flattening may sometimes very positively exist, but, from being unequally shared by both sides; escape detection: when present to any extent, the clavicle is thrown into unnatural relief.

Vocal vibration is certainly increased in intensity under the clavicle; but the amount of increase is trifling. Its existence is, therefore, with difficulty established under the right clavicle, where there is a natural excess of fremitus; and on the left side, as a sign of incipient tuberculisation, increased fremitus commonly fails us, because priority of disease on that side is most common in females, who, unfortunately, from the quality of their voices, have naturally little or no vocal vibration.

Careful admeasurement with the callipers will sometimes detect positive reduction in antero-posterior diameter at this period. If the deficiency be marked, pleural false membrane is probably present to some amount. The motions of the infra-clavicular region are perverted, to the eye, to the hand, and to the measure. The hand laid flat on the surface, instead of being arched outwards during inspiration, is simply raised upwards; elevation-movement exists, expansion-movement is wanting; nay, more, the infra-clavicular region may actually sink in with inspiration. The ribs, if the pleuræ are agglutinated, may be *felt* to converge at the same time. The deficiency of expansion-movement under the clavicle is a more valuable sign in the female than in the male, for the obvious reason that it is naturally much more limited in the latter than in the former.

The results of percussion may or may not be significant. Slightly impaired clearness of sound, and slightly increased parietal resistance, may be produced by a very few scattered tubercles at the apex,—local collapses of the lung doubtless contributing to impair the resonance. The deficiency will be earliest caught at the inner aspect of the supra-clavicular and clavicular regions (see Diagrams I. and II.), where the apex of the lung lies. To detect it, percussion must be very lightly made, and, for purposes of certainty, repeated in various postures both of the patient and of the percussor. Care must be taken to direct the percussion *from*, and not *towards*, the trachea: with this caution, percussion is much more conclusive at the inner than the outer part of the supra-clavicular region. Slight dulness is a more valuable sign in the female than in the male, and more valuable in both sexes at the left than the right side (p. 53). Habitually, a greater superficial area of disease is required to affect the percussion-sound in the infra-clavicular and supra-scapular regions; but occasionally even the latter suffers before the front of the chest. Neither in front, nor behind, does the dulness extend beyond the middle line. Sometimes, when tubercles are sparingly scattered through an entire apex, percussion on a broad surface will disclose a difference in resonance, imperceptible when a single finger is used as the pleximeter. Or recourse may be had to the dynamic tests: the increase of clearness produced by a full inspiration will be, in comparison with that on the healthy side, very trifling; and, on the other hand, there will be comparatively a great diminution of clearness at the close of a complete expiration. As deposition, collapse, and consolidation advance, the sound becomes duller and duller, but never becomes totally toneless, or of putty-like flatness. If consolidation extend across from the costal surface to the trachea, or large bronchi, more especially if the pleural laminæ be agglutinated at the spot, the percussion-sound may be wooden, or even tubular. The fallacy of co-existent emphysema is always more or less to be apprehended during this stage. The respiration in the infra-clavicular region, and also usually in the upper scapular, is affected in *intensity*, being weak and almost suppressed in some points, exaggerated in others; perverted in *rhythm*, being frequently jerking; impaired in *quality*, being harsh, bronchial, or even slightly blowing. The value of these states of respiration is directly as the limitation of the area within which they are discernible; if they exist above, and are imperceptible below, the second interspace, they are very seriously significant. Slight harshness of respiration is more valuable, as a sign, in the male than in the female, and notably so on the left than on the right side. If the other causes of jerking rhythm (p. 87) can be excluded, which may or may not be difficult, this condition of rhythm, when limited to one apex (it is rarer posteriorly than anteriorly) becomes a really important sign of tuberculisation. My opinion on this point has yearly grown more positive. It is to be remembered that it occurs at a period of the disease when the physical signs generally are few in number, not so decisive as might be wished, and when, of course, every addition to their number is really important. According to my experience, it is a persistent phenomenon. I have not, as Dr. Stokes appears to have done, succeeded in removing it by local treatment, even in cases where other signs, *e. g.* harshness of respiration, were favourably modified by that treatment. It may exist in the highest degree without any co-existent affection of the pleura; and must not be confounded with grazing friction-sound. Prolonged expiration, if unattended with alteration of quality, is insignificant: under such conditions, it is in all probability a normal state; and, even coupled with slight harshness and coarseness of quality, it must be cautiously received as evidence in females, and at the right side.

The only rhonchal sound specially belonging to this stage of phthisis is the dry crackling; occurring towards the close, it gradually passes (with the exceptions already referred to) into the humid crackling variety. Occasionally at the close of this stage the peculiar condition, I have designated as *cogged-wheel rhythm* of respiration, exists, oftener in the supra-spinata fossa, than below the clavicle: I have *never* met with this condition, except in tuberculous patients.

The vocal resonance varies to such an extent in amount and quality, as to make it totally unworthy of clinical confidence: I have known it (where the existence of consolidation was positive, either from other signs, or, in addition, from *post-mortem* examination) of the average characters of health, weak, null, exaggerated, intensely bronchophonic, or pectoriloquous. The state of vocal fremitus, curiously enough, does not vary thus, and is hence, comparatively, a more trustworthy guide: but its positive value, we have seen, is next to nothing. The reasons why vocal resonance should thus vary are amply explained in the First Part of this work.

In a doubtful case of tuberculisation of the right apex, if the heart's sounds, but especially the first, be more clearly audible near the clavicle on that side than the left, we have a point of evidence of consolidation; but the absence of this sign will not disprove the existence of solidity, of which strong evidence appears on other grounds. Subclavian murmur may be present; and also systolic murmur at the second left, or pulmonary, cartilage, associated or not with basic systolic, or subclavian, murmur. To the murmur, pulmonary in site, Dr. Latham attaches much importance, as an attendant on tuberculisation,—but I confess its diagnostic claims do not appear to me to be established: as to its frequent absence, I take it for granted that so positive a fact must be admitted on all hands.

The signs of dry or plastic pleurisy, and of bronchitis and of pneumonia, may occur in connection with tuberculous deposition; they have indirect value, if limited to one or both apices. Tuberculous patients, the *apex* of whose lung is the seat of capillary bronchitis, are of course liable to be seized with idiopathic bronchial inflammation of both *bases* from accidental causes. Under such circumstances, it is curious and interesting to observe the manner in which the upper and lower rhonchi travel towards each other so as eventually, in some rare instances, actually to meet towards the middle height of the lung. Where such union of the rhonchi takes place, the case is of the most serious character. I may further observe, with respect to the symptomatic rhonchi occurring in connection with tubercle at the apices of the lungs, that I have found true crepitation, to say the least, singularly rare; that is, unless in cases where the cause of the rhonchus is really extensive pneumonia,—such pneumonia as shall during its existence assume, in point of importance, all the characters of the idiopathic inflammation. Under the ordinary circumstances of acute irritation setting up in the neighbourhood of the new matter, the subcrepitant is the rhonchus audible, and capillary bronchitis the anatomical state present.

II. The signs of the *second stage*, that of softening, are in part new, in part those of the first stage, either stationary or carried to a higher point. To begin with the latter: depression, both above and below the clavicle, is now greatly more marked, and may sometimes be really present to a notable amount, but be masked by twisting of the clavicle downwards and inwards on its long axis. When the clavicle thus, as it were, follows the retreating ribs, the callipers, or chest-measurer, supply the only trust-worthy evidence as to the amount of depression. The corresponding supra-scapular region, if one apex be more affected than the other, is distinctly hollower than its fellow,—a fact apparently so strange, that nothing but repeated observation would justify its statement. The vocal vibration does not increase as a consequence of the softening process; but as this is, generally speaking, accompanied with extension of induration also, such increase may occur. The semi-circular measurement of the side, and the transverse diameter of the chest in the axillæ, are lessened in consequence of the general deposition of tuberculous matter, atrophy, and interstitial contraction of the lung, together with, in some cases, contraction of pleural false membranes. Dulness under percussion gains in area and in intensity, and is, oftener than in the first stage, wooden or tubular. The respiration grows more extensively and markedly blowing, or remains merely bronchial. Vocal resonance varies as before. The *new* phenomena in this stage are humid crackling, and thin metallic mucous, or large subcrepitant, rhonchus. When elimination of the softened material commences, the rhonchus may become cavernulous.

In consequence of the diminution of the mass of the lung, the heart may be elevated above its natural position, the diaphragm raised, and the mediastinum drawn towards the mainly affected side. The heart's size gradually diminishes; but its area of superficial dulness may be apparently increased, in consequence of contraction of either, but especially of the left, lung.

III. The signs derived from inspection in the excavation-stage remain as previously. I have, however, in some rare instances, known extreme infra-clavicular depression of the second stage diminish somewhat, nay, even slight bulging occur, when a *capacious* excavation had formed. This will probably occur where the anterior wall of the excavation is excessively thin, indeed merely membranous; the condition becomes one, physically speaking, closely assimilable to local pneumo-thorax: at all events, of the fact I am positive. Again, in such cases, inspiratory expansion may improve to a certain extent. I have known this in a case where amphoric respiration and cracked-metal percussion-sound gave evidence of the size of the excavation; and the more solidified the rest of the lung, the greater will be the expansion over the cavity.* Rhonchal fluctuation may sometimes be detected; and if the cavity be of large dimensions, fluctuation, produced by succussion of the trunk, may be felt. The percussion-results depend less on the fact of cavity existing than on the conditions of the cavity. If it be small, and surrounded with much indurated lung, the sound will be absolutely dull, or wooden, or tubular. If there chance to be a thick stratum of sound tissue between the excavation and the ribs, gentle percussion may be of almost healthy quality,—moderate dulness, slightly tubular, too, is detected on strong percussion. If there be several small excavations, with indurated substance between them, the sound is markedly dull, and somewhat tubular; if one or two large excavations, amphoric or of cracked-metal quality.

The respiration, provided the cavity be neither distant from the surface, nor separated from it by a stratum of healthy tissue, is hollow, divided, hoarse, blowing, and cavernous, or actually amphoric; and this state alternates or co-exists, in the manner already described (p. 96), with gurgling rhonchus. The cough is cavernous or amphoric; and metallic echo or metallic tinkling in some rare cases accompanies the voice, cough, and respiration. The vocal resonance varies in characters; it may be pectoriloquous;

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* Green, U. C. H., Females, July, 1850. Expansion is very rarely totally deficient over a cavity; greatly more rarely than over highly consolidated texture.

amphoric, bronchophonic, natural, weak, or null. The conditions of these varieties, in part understood (p. 133), are in part unintelligible; and hence vocal resonance should never be trusted to alone in the diagnosis of a cavity. The sole form of resonance that can be held distinctive of an excavation, is *whispering* pectoriloquy; but cavities may exist without this.

A cavity of large size, with hard and smoothish walls, and containing thin fluid in moderate quantity, may emit a guggling sound when the trunk is abruptly shaken. The heart's action, too, (if the cavity be near) produces a similar sound of cardiac rhythm.

Where great loss of substance is produced in the right lung by excavation, and its remaining tissue is much reduced in bulk, the heart may be drawn greatly out of its place, and beat to the right of the sternum.

IV. The physical signs of *arrested phthisis* have never yet been systematically examined. And as these signs must vary not only with the stage and conditions of the disease actually present at the moment suspension of its progress was effected, but also with the length of time that has intervened between such suspension and examination of the chest, it is extremely difficult to establish any general rules on the subject. I shall consequently simply put down a few specimens of the conditions that have fallen under my notice in particular cases.

(*a*) Notable depression, supra- and infra-clavicular and suprascapular, imperfect expansion, very weak, harsh respiration, dulness under percussion, exaggerated fremitus, and strong vocal resonance. Here softening signs on a small scale had existed eighteen months before.

(*b*) Similar depression, imperfect movement and dulness, dry clicks, with deep inspiration, which is weak and harsh. Here softening signs had existed only six months before.

(*c*) Dulness under percussion at the apex, feeble bronchial breathing, with a deep-seated creaking sound in inspiration (p. 113.) Here, nine months before, softening signs were actively present, and both the local and general symptoms seemed to promise a rapidly fatal issue. I have not seen this patient professionally since the above observation was made, and cannot say what the local state may now be (seven months later); but I know by report that her general health is excellent, and that there are no chest symptoms, except occasionally slight cough.

(*d*) Respiration of laboured jerking rhythm, weak and bronchial, but without clicks or rhonchal sound of any kind, and with very slightly deficient resonance under percussion. Here there had been, two months earlier, much greater dulness under percussion, respiration of diffused blowing type and cogged-wheel rhythm,—and the general symptoms of advancing tuberculisation.

(*e*) Dulness under percussion below the clavicle, with slightly tubular quality, hollow dry cavernous respiration, vocal resonance diffusely exaggerated. I have to-day seen a case of this kind, where, from the account given me, I infer that four months ago the progress of phthisis was most active. At a later period the same signs would be found; for there is no evidence, that I know of, positively showing that the walls of an excavation of any size (of a size, for example, sufficient to give cavernous signs) may actually cohere, in other words cicatrise. But the patient may under these circumstances be practically as free from complaint, as if actual cicatrisation had occurred.*

(*f*) I have known some instances where the physical signs of induration had existed at either apex to a slight amount, where the local and general symptoms of phthisis had made their appearance in individuals belonging to a tainted family, and where the signs in question totally disappeared along with the symptoms, leaving behind them merely trivial harshness in the respiratory sounds. These I believe to have been examples of phthisis arrested in the first stage; but the positive demonstration of the fact I admit it would be difficult to furnish.

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* *Vide* Cyclop. of Anat. and Physiol. Art. Adventitious Products, p. 109.

V. The *symptoms* of phthisis do not admit of such systematic division into stages as its physical signs: there is no symptom absolutely peculiar to any one of the three stages. It will be better, therefore, to consider them one by one under the heads of *local*, *general*, and *incidental*.

A. *Local.* Cough, unless in latent cases, is an essential symptom; habitually unpreceded by coryza, rarely paroxysmal; it is at first either dry (but very probably in not more than about one-tenth of cases) or attended with colourless, frothy, watery, or mucilaginous-looking expectoration, the last somewhat characteristic. The thinner fluid at an early period sometimes deposits a thicker, grumous-looking substance in small quantity, resembling the deposit in barley-water: I do not believe that this occurs except in phthisis. Gradually the expectoration becomes glairy and mucous, striæ of opaque buff or pale yellow appearing on a lighter ground; it grows less and less aërated: presently the sputa become purulent, either in the form of small pellets with sharply cut outlines, opaque, semi-floating, non-aërated, and of yellowish colour (boiled-rice sputum), or of larger masses with ragged outlines, or of broad, flat, discoloured lumps, darkish green in tint, but smooth in outline, and remaining apart if expectorated into water. Eventually the sputa acquire an ash colour, run together in one mass, totally free from air, and are constituted essentially of pure pus: this condition of sputa is of very bad augury, if it occur in a patient under treatment: when found in patients who have greatly neglected themselves up to the time of seeking advice, it is generally rapidly modified by medical care. Occasionally sudden profuse expectoration of purulent fluid occurs, in some cases traceable to abrupt evacuation of a cavity, in others to abundant secretion from the walls of old cavities and neighbouring bronchi. The quantity of expectoration varies greatly in a mass of cases: sometimes profuse, especially in the earlier and closing periods, it may be very slight throughout; and I have known cases run their course without any expectoration at all. I once saw a lad, aged fifteen, admitted into hospital with typhoid fever, become tuberculous during convalescence, and, dying within a short period, present large cavities in his lungs, though he had never, as far as could be learned by constant questioning, expectorated a single sputum: he must, of course, have swallowed them all, as *children* invariably do.

Hæmoptysis, including under this term even expectoration simply streaked with blood, is a symptom of extreme frequency, occurring, as I formerly found,* in about 81 per ·100 of cases.

The following general inferences are derived from the examination of 106 cases:—

> "Hæmoptysis was of very slightly (4 per 100) more frequent occurrence in males than females.—Hæmoptysis to a medium amount is about four times less common than to very slight or to profuse amounts (both taken together). Very profuse hæmorrhage from the lungs is more common in males than females.—Medium frequency of recurrence of hæmoptysis is materially less common (and this in both sexes) than a single or than repeated attacks. Further, repetition of hæmoptysis is more common in males than in females. —It is materially more common for a first hæmorrhage to be more profuse than subsequent ones, than for subsequent ones to be more profuse than the first.—Hæmoptysis is more frequently met with (and this independently of any influence of duration of the disease) in persons who have reached the second and third stages, than in those whose lungs have not yet softened. This proposition is more markedly true of males than of females.—There does not appear to be any notably greater tendency to hæmoptysis, where the right lung has reached a more advanced stage than the left, nor *vice versâ*, where the left has taken the lead. It seems improbable that either lung is more effective in causing hæmoptysis than its fellow.—The frequency of hæmoptysis increases with advancing years in both sexes. The increase is more abrupt in females than in males, and in the former appears connected with the catamenial function.—This greater frequency of hæmoptysis in persons of more advanced years, does not depend altogether on greater duration of the disease; for those who had had hæmoptysis, had been phthisical for only a mean period of eight months longer than those who had not spit blood.—The most common periods for the occurrence of hæmoptysis, were, at the very outset, or after the expiration of the first month; it is very rare for hæmoptysis to occur within the first month, unless it has actually appeared as the first, or among the first symptoms. The phrase "first symptom" here is to be understood with a qualification to be by-and-bye explained.—In upwards of half the cases of notable hæmorrhage

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(beyond 4 oz.) this occurs, or has occurred, as the "first symptom," corroborating the inference as to the excess of amount of first over subsequent hæmorrhages.—Hæmorrhage of this amount is rare as a coexistence with other first symptoms (in $\frac{1}{13}$ of these cases) appreciable by the patient.—Streaked or tinged sputa are, on the contrary, of very common appearance amongst the earliest symptoms.—But streaked or tinged sputa are rarely (or never) *the* "first symptom" singly and alone.—Season does not appear to exercise any marked influence on the occurrence of a first hæmorrhagic attack.—Hæmoptysis never appeared as the *bonâ fide* first symptom in these cases, the phrase being understood in its absolute sense without qualification; it is so only in the sense that it is the first symptom particularly noticed by patients,—the first occurrence that leads them to watch their health."

The last proposition is important: it leads one to reject, on clinical grounds, the old notion, that phthisis is ever caused by hæmoptysis in those cases where it *appears* to lead the way in the train of morbid events ("phthisis ab hæmoptoë"). The quantity of blood brought up varies between a few streaks and some pints of pure unmixed blood. Hæmoptysis may kill *directly* or *indirectly*. My series of 131 cases of phthisis furnishes but two examples of such mode of death. In one, death was direct, but from asphyxia, not loss of blood: the trachea and bronchi, as far as traceable, were plugged with coagula. In the other, death occurred from exhaustion five days after the hæmorrhage. In these and a few other cases that have fallen within my observation, the patients have invariably been *males*; in this point of view, and in some others, hæmoptysis is a more serious event in men than in women. A first hæmorrhage having been severe, it is unlikely that a subsequent one will kill *directly*; for it is not often that a subsequent hæmoptysis is more severe than a first severe one, granting that they do not follow so closely upon each other as to appear a mere continuation of one and the same attack. One of the cases above referred to constituted an exception to this rule. It appears from the cases I have examined, that hæmoptysis is frequent in proportion to the duration of the primary disease; if so, the converse of the proposition cannot be evaded; and we are forced to conclude that *frequently-recurring hæmoptysis does* *not reduce the mean duration of life, after seizure with tuberculous symptoms, in any given mass of cases.* It is not repugnant to reason to admit, then, that in a certain number of instances hæmorrhage from the lungs may act as a local therapeutical agent. No doubt the disease sometimes runs on more rapidly after an attack of hæmoptysis; but I cannot help regarding the two things as mere coincidences. In the course of phthisis, whether in a quiescent or active state, calcareous particles of some size may be expectorated. I have known this continue for years.

The cough in phthisis more frequently brings on vomiting or nausea, than in any other pulmonary affection: if a fit of coughing occurs after a meal has been taken, more or less of it is habitually vomited in the majority of cases that have reached the second stage.

Dyspnœa is the reverse of a prominent symptom. The frequency of respiration is rarely carried to any notable height in the pure disease; and when the act is morbidly accelerated, the pulse also is so much quickened, that the ratio of the two is scarcely affected. I have seen few cases of very marked dyspnœa, of which the patients volunteered complaints. In these cases there was either great emphysema, with bluish lividity of the face, and general coolness of surface, or nothing could be detected except the tuberculous disease to account for the symptom.

Actual pain, or distressing sensations within the thorax, exists in the great majority of cases; but severe suffering of this kind is the exception. The pain may in part be intra-pulmonary, true pleurodynic, neuralgic (intercostal), or pleuritic. The last is by far its most frequent cause. To local evanescent dry pleurisies are in the main due the wandering chest-pains of these patients, and to more permanent pleuritic changes their comparatively fixed sufferings.

B. *General.*—Febrile action sometimes attends the very earliest local symptoms,—in sixteen of ninety patients observed by Louis, excluding cases of acute phthisis. In the majority of instances, it does not appear till softening has occurred. Actual shivering, or merely chilly sensations, with subsequent heat, followed by perspiration, constitute a complete paroxysm. The shivering is slight, as a rule,—the perspiration profuse. The chest and head are in many cases the chief seats of perspiration. Sudamina are not common. There may be two daily paroxysms,—a morning and an evening one.

The pulse is habitually, but not invariably, frequent; the range is wide; within my own observation from 60 to 140 per minute in uncomplicated cases. The frequency of the pulse sometimes changes remarkably in the same case within a day or two; occasionally an obvious cause, in the shape of some intercurrent attack, can be found for this,—quite as commonly it baffles explanation. It has been said that the variation of frequency in the lying and standing postures is less than in other diseases attended with debility. I have repeatedly met with marked exceptions to the statement.

The digestive organs suffer more or less in the majority of cases. Thirst is rarely absent through the whole course of the disease, whether the alimentary canal be free from anatomical change or not. Pain and tenderness below the ensiform cartilage, with loss of appetite, nausea, and occasional vomiting, indicate morbid change in the mucous membrane of the stomach, and can scarcely be considered direct symptoms of the primary disease. Vomiting of food, brought on by fits of coughing, and unaccompanied by any other gastric symptom, does not indicate any textural change in the organ.

Stomatitis (diphtheritic or not) is not uncommon towards the close of the disease; epiphytic formation occurs, but rarely, in the exudation.

The state of the bowels varies greatly,—in some rare cases natural, in a certain number constipated even to the last hour, in others relaxed from time to time, in others permanently loose. Diarrhœa may depend merely on secretive changes in the bowels, on small ulcerations in the small intestine, or on extensive destruction of the mucous membrane of the colon, as well as the ileum. In the first case, the symptom is temporary, and easily controlled; in the second, more persistent, and with difficulty arrested; in the third case, absolutely unmanageable. In the last case, as insisted on by M. Louis, the motions are very numerous, reddish, or putty-coloured, fluid, and of putrid odour. If the rectum be ulcerated, the motions may be pseudo-dysenteric.

The cellular tissue remains remarkably free, as a rule, from serous infiltration. If there be marked œdema even of the ankles, there is generally some morbid state, besides phthisis, to account for the fact.

The mental faculties retain their clearness in the majority of cases to the last few hours—trifling failure of memory, and inability to follow a train of thought, alone existing towards the close. Slight wandering at night, on waking out of sleep, sometimes occurs. When marked cerebral symptoms exist, they always indicate intercurrent disease. The temper, though irritable, is singularly hopeful. Every one has seen cases in which arrangements for future years are made within a few days of death; and I have actually known the question of a change of profession complacently considered within *three hours* of the fatal event. There may be, in such cases, an effort on the part of patients to deceive themselves and those about them, as to the real state of things; but, nevertheless, hopefulness constitutes a special clinical feature of the disease, and cannot by any means always be explained by the absence of pain. Contrast the phthisical with the cancerous patient in this point of view.

Emaciation is one of the essential phenomena of phthisis. Preceding, as it does, in some cases all other symptoms, local or general, it is obvious that the presence of tubercles in the lungs acts as an efficient cause of wasting,—that loss of appetite, vomiting, diarrhœa, and perspirations, are subsidiary in their influence. It is not only the external fat and cellular tissue (those of the face, often, least) that waste; the muscles, and the parenchymata suffer too; as proved by Louis and Bizot, the weight of the heart and calibre of the aorta are less in the victims of phthisis than of any other disease, except cancer. The weight of the body, as a whole, consequently diminishes, rapidly, surely, and progressively, more especially as no serous accumulations form to give fictitious weight. But there is a curious fact connected with the emaciation of phthisis (and, for aught I know to the contrary, of other chronic diseases); namely, that it is not an invariably steady process. I have repeatedly found, by weighing patients within short intervals, that there are rises and falls in weight, intercurrent to the general progressive tendency downwards, and occurring irrespectively of any obvious changes in diet, appetite, or colliquative symptoms. The late Dr. Robert Williams, of St. Thomas's, as I several years ago learned from his friend, Dr. Silvester, inferred from a large mass of observations upon this point, that there was a law of periodicity regulating the rises and falls of weight. I have no means of ascertaining what the period established by Dr. Williams was; and my own observations are too limited to supply the deficiency. I feel tolerably sure that the interval is shorter than a month—the period at which Sanctorius found a passing increase of weight took place in health.

M. Louis has arrived at the conclusion, contrary to what had previously been held, that the sexual appetite in the male undergoes impairment. Menstruation, he found, ceased in the female, when the total duration of the disease was under a year, at about the ninth month; when of longer duration, at a relatively later period. Menstruation may continue to the last. It has been generally supposed that pregnancy retards the progress of phthisis,—the disease acquiring increased activity after parturition. I have positively observed several cases in which many of the symptoms of phthisis became less prominent during pregnancy. This might be the fact, it is true, without the real pulmonary disease being suspended in progress; but it is curious that I do not remember to have opened, or to have seen opened, a female dying of phthisis and at the same time pregnant.

My sphere of observation is, however, not the most favourable for encountering pregnant women. M. Grisolle has lately examined these questions, and comes to the conclusion that the disease is somewhat increased in rapidity of progress during pregnancy; while, after parturition, it is slightly mitigated, or at least, remains stationary. The number of cases he has collected (and all other persons that I know of are in the same predicament) are quite insufficient to solve the problem.

I some time since numerically examined the question, whether the tuberculous diathesis intensifies or weakens the force of fecundity in the female, and of the procreative faculty in the male, and was led, by the facts, to the conclusion, *that the procreative power of phthisical males is below the average—the fecundity of phthisical females materially above it.* Taking the two sexes together, and regarding them as phthisical stock prepared to propagate, the female activity is counter-balanced to a certain extent by the male inactivity; 11·82 years of phthisical cohabitation produced a mean of 0·83 children less than 17·48 years of non-phthisical cohabitation.*

The blood in the early period of phthisis is deficient somewhat in red corpuscles, and very slightly in fibrine; the proportions of albumen and water are increased; the serum appears to be less alkaline than in health. With the advance of the disease the blood becomes hyperinotic, from the irritating influence of the tubercles and intercurrent inflammations. Quite at the close the fibrine and the solids generally undergo notable diminution. There is no microscopical character, that I know of, in the red corpuscles, which can be trusted to as the slightest guide in the diagnosis of the disease.

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* For the facts themselves, *vide* Medical Times, July 6, 1850. The above results accord with M. Louis's general statement, concerning the failure of sexual inclination in the male; they are totally at variance with an assumption of M. Grisolle, that conception is rare in phthisical women. The total number of cases on which my inferences are founded, is 91 of phthisical, 220 of non-phthisical, persons.

In the early stages the urine presents nothing remarkable; as inflammations occur, it grows febrile; it may become anæmic from hæmoptysis, diarrhœa, &c. It contains an excess of uric acid as a rule; occasionally minute quantities of sugar;* sometimes it is passingly and slightly albuminous. The existence of sugar is interesting in connection with the well-known tendency of diabetes to terminate in phthisis, and also as subversive of the hyper-oxidation theory of the latter disease.

C. The *incidental* symptoms of phthisis are the clinical expressions of its "secondary morbid changes." A case of phthisis may run its course without a single one of these ever occurring, or they may occur in various combinations and variable intensity. Sometimes these symptoms are so severe as to throw into the shade those of the pulmonary disease, and monopolise the attention both of patient and practitioner; while the secondary changes causing them, sometimes seem to accelerate, sometimes retard, the advance of the primary affection, and prove themselves the real causes of death.

(*a*) Pneumonia frequently occurs in the course of tuberculous disease, or at its close. In the former case, it is either a mere local effect of the progress of tuberculisation, or it may be extensive, and acquire almost the importance of an idiopathic attack of the disease. But even then it is rarely of serious augury; it is, singularly enough, less fatal than primary pneumonia. What I have observed on this head, is in perfect accordance with the observations of Louis;—the mean duration of the inflammation, even, is less than when occurring in sound lungs. Some of the most marked examples of rapid resolution I have met with, were in phthisical persons. No satisfactory proof has ever been given that intercurrent pneumonia accelerates the progress of the tuberculous disease itself.

M. Louis holds that pneumonia occurring at the closing periods of phthisis, is almost of necessity fatal. But, admitting this, it does not follow that there is any special tendency in the phthisical to death by pneumonia. In point of fact, pneumonia—or conditions of the lung referred to that disease—is a tolerably common appearance in the lungs of persons cut off with all varieties of chronic maladies. The proportion of cases in phthisis is scarcely greater; and where so-called hepatisation is found, it has not always been the actual cause of death.

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* *E. g.* Case of Eliz. Arthur, U. C. H., Females, vol. vi., p. 51. Sp. gr. = 1024.

Pneumonia limited to the *anterior* portion of either apex, is, in the great majority of cases, tuberculous,—not invariably so, however. I have known pneumonic signs limited to the infraclavicular region, without being the result of tuberculous irritation, so far as the disappearance of all vestige of morbid signs at the apex can be accepted as proof of this.

Pleurisy we have already met with in the dry and exudation forms, as an attendant on tubercle. Effusion occurs in a fair proportion of cases, and is always a most serious complication: complete recovery is singularly rare. Double pleuritic effusion is occasionally met with, and is, as shown by Louis, almost peculiar to phthisis. Effusion, single or double, hastens the fatal issue.

Bronchitis, local or general, invariably occurs in the course of phthisis. The form most peculiar to the disease seems to be that limited to one apex or to one base.

Ulcerations of the epiglottis give rise to great dysphagia, especially of liquids, which frequently return by the nostrils. There is fixed pain in the throat, opposite the affected part.

Chronic inflammatory changes in the larynx are indicated by change in quality of the voice and cough, which grow hoarse, muffled, and cracked; and in proportion as ulceration destroys the chordæ vocales, the voice degenerates into a hoarse whisper. I have never observed absolute aphonia. Pain, stinging, pricking, or shooting, is more or less constant; and a distressing sensation of dryness is experienced. If the epiglottis be free, there is no dysphagia. The physical signs are rough, coarse respiration in the larynx, with sonorous, sibilant, or thin gurgling rhonchus, according to the dryness or moisture of the diseased surfaces.

I know of no positive symptoms of ulceration of the bronchi; those of chronic tracheitis are obscure. Pain, heat, and dryness, with choking sensation above the sternal notch, are all that I have observed; and these symptoms may exist without tracheal ulcers, while ulcers may exist without them. Intense, so-called tracheal dyspnœa, has not fallen under my notice.

The symptoms of perforation of the pleura have already been described.

(*b*) Of abdominal incidental symptoms, those indicating chronic peritonitis are the most important. Enlargement of the abdomen, pain and tenderness under pressure, ascites, and tympanitic distension of the intestines, are the prominent symptoms. The ascites may rapidly disappear, the tympanitis remains, and the outline of the intestines appears on the abdominal wall. Pain may be constant, or occur only at the moments flatus moves in the bowels. All control over escape of flatus may be lost; probably, by a consensual act for the avoidance of pain, the muscular effort to retain it is omitted. Diarrhœa is present in the majority of cases, whether the bowel be ulcerated or not. This secondary affection may fall into a quiescent state; but if so, the pulmonary disease unfailingly, or almost unfailingly, grows more active.

Fatty disease of the liver, not a common secondary change in this country, has no special symptom, that I know of. Probably it affects the properties of the fæces. Its physical signs are those of simple enlargement of the organ.

Fistula in ano is, according to my observation, more frequently met with (more in males than in females) than it has of late been the habit to believe.

Serious as are the evils of ulcerations of the bowels, those of cicatrisation may be more so. Death may be the result of accompanying contraction of the bowel. M. Louis reports a case, where symptoms of intestinal stricture, the earliest observed in its course, proved fatal in about twenty-two months, having throughout kept the chest-symptoms in abeyance, though cavities existed in the lungs.

(*c*) The only cerebral affection of importance,—tuberculous meningitis,—has the following symptoms:—Cephalalgia (frontal) and vomiting, flushing and pallor of the face, followed by delirium, commonly of the quiet kind, very rarely boisterous or violent, paralysis, hemiplegic or partial, twitchings of the face, strabismus, somnolence, and eventually coma; the pupils, at first commonly contracted, eventually dilate; the pulse and respiration both slacken; the skin becomes cool; the cough and other chest-symptoms disappear. Remission of the cerebral symptoms, simulating convalescence (though I have never seen it so marked in the adult as in the child), may occur towards the fatal issue, which generally takes place in from seven to eighteen days. Whether recovery is possible, will be considered hereafter. I have now observed at least six cases of this affection in the adult, in which a peculiar form of mutism formed a striking symptom. The patients, when questioned, looked steadily in the speaker's face for a few moments, and then, without making the slightest effort at speech, deliberately, but without any sign of petulance, turned their heads away.

Deafness, depending on tuberculous destruction of the membrana tympani, is noticed in some cases.

VI. Chronic phthisis may, especially in females, for a portion or for the entire of its course, whether this be of medium or considerable duration, run a *latent* course. That is, tubercles may exist in the lungs and slowly work out their ill influences on the organism, though secondary blood changes, without awakening attention by any of their ordinary local subjective symptoms, such as cough, expectoration, pain in the chest, and dyspnœal sensations.

Four classes of cases may be met with referrible to this head. To the first belong instances in which violent hæmoptysis, or perforation of the pleura, are apparently the first symptoms of the disease,—the former commonly so, the latter very rarely so. To the second belong cases of slow course in which one of the secondary morbid states, such as chronic peritonitis, or ulcerative diarrhœa, mask or really suspend the progress of primary mischief. In a third class we find cases, where an individual is generally out of health, without suffering from local or general symptoms of any severity. In a fourth rank cases where very prominent symptoms exist, such as emaciation, fever, loss of appetite and sleep, occasional relaxation of the bowels without apparent cause,—none of them of obvious pectoral origin.

The local latency of tuberculous disease in some of these cases seems explicable on the principle of *Duobus* morbis *simul obortis, vehementior obscurat alterum.* But in instances where none of the secondary morbid states exist, the fact baffles explanation. In some of these latent cases the fever is exceedingly marked. But the great points for the observer to bear in mind are, that, while such latency is not only a real, but a frequent, clinical fact, physical signs alone can disclose the true state of things. A single tap above the clavicle will sometimes give the ready clue to what has hitherto been utterly mysterious. Let him not be diverted from his belief by the assurances of patients that they have never coughed,—the assurance will occasionally be given by persons who, at the moment they give it, have *cavities in their lungs.*

VII. I propose, instead of systematically considering the *diagnosis* of chronic phthisis, to embody in a series of propositions, some of the most interesting facts, of which I have actually seen illustrations.

(*a*) A young adult, who has had an obstinate cough, which commenced without coryza and without any very obvious cause, a cough at first dry and subsequently attended for a time with watery or mucilaginous looking expectoration, and who has wandering pains about the chest, and loses flesh even slightly, is in all probability phthisical. (*b*) If there be hæmoptysis to the amount of a drachm even, the diagnosis becomes, if the patient be a male and positively free from aneurism and mitral disease, almost positive. (*c*) If, in addition, there be slight dulness under percussion at one apex, with jerking or divided and harsh respiration, while the resonance at the sternal notch is natural, the diagnosis of the first stage of phthisis becomes next to absolutely certain. (*d*) But not absolutely certain: for I have known every one of the conditions in *a*, *b*, and *c* exist (except hæmoptysis, the deficiency of which was purely accidental), when one apex was infiltrated with encephaloid cancer, and no cancer had been discovered elsewhere to suggest to the physician its presence in the lung. (*e*) If there be cough, such as described, and permanent weakness and hoarseness of the voice, the chances are very strong (provided he be non-syphilitic) that the patient is phthisical. (*f*) If decidedly harsh respiration exist at the left apex or at the right apex behind, if the rhythm of the act be such as I have called *cogged-wheel*, and there be dulness, so slight even as to require the dynamic test for its discovery, there can be little doubt of the existence of phthisis. (*g*) If with the same combination of circumstances deep inspiration evokes a few clicks of dry crackling rhonchus, the diagnosis of phthisis, so far as I have observed, is absolutely certain. (*h*) If these clicks on subsequent examination grow more liquid, the transition from the first to the second stage may be positively announced. (*i*) If there be slight flattening under one clavicle, with deficiency of expansion-movement, harsh respiration and slight dulness under percussion, without the local or general symptoms of phthisis, the first stage of tuberculization cannot be diagnosticated with any surety, unless there be incipient signs at the other apex also: the conditions in question limited to one side might depend on chronic pneumonia or on thick induration-matter in the pleura. (*k*) The existence of limited, though marked, dulness under one clavicle, with bronchial respiration and pectoriloquy, so powerful as to be painful to the ear, the other apex giving natural results, will not justify the diagnosis of phthisis. I have known this combination when the apex of the lung was of model health, and a fibrous mass, the size of a walnut, lay between the two laminæ of the pleura. I would even go further, and say that the combination in question is rather hostile than otherwise to the admission of phthisis; as, had tuberculous excavation formed at one side, the other lung would, in infinite probability, have been affected in an earlier stage. (*l*) Pneumonia, limited to the supra- and infra-clavicular region on one side, and not extending backwards, is commonly, but not always, tuberculous. (*m*) Subcrepitant rhonchus, limited to one base posteriorly, is not, as has been said, peculiar to tubercle; it may exist in emphysema and in mitral disease. (*n*) Chronic peritonitis, in a person aged more than fifteen years, provided cancer can be excluded, involves as a necessity the existence of tubercles in the lungs. To this law of Louis's, it is necessary to add the qualification, provided Bright's disease be also absent. (*o*) Pleurisy with effusion, which runs a chronic course in spite of ordinary treatment, is, in the majority of cases, tuberculous or cancerous: the character of the symptoms, previously to the pleurisy, will generally decide between the two. (*p*) Double pleurisy, with effusion, is not, as has been said, significant of tubercle; for it may depend on Bright's disease. If the latter disease can be excluded, carcinoma and pyohæmia remain as other possible causes. (*q*) If a young adult, free from dysentery, and who has not resided in tropical climates, suffers from obstinate diarrhœa, which goes on month after month, with slight remissions or intermissions, even though there be no cough, he is in most strong probability phthisical. If physical signs, to the slightest amount, exist at either apex, he is, almost to absolute certainty, phthisical. (*r*) If a young adult, free from secondary syphilis and spermatorrhæa, and not dissolute in his habits, steadily lose weight, without clear cause, he is in all probability phthisical, even though no subjective chest-symptoms exist. (*s*) But he is not by any means certainly so; for he may have latent cancer in some unimportant organ, or he may have chronic pneumonia. (*t*) Nay, more, he may steadily lose weight, have dry cough, occasional diarrhœa, and night-sweats, and present dulness under percussion, and bronchial respiration under both clavicles, and yet be non-phthisical. I have known all this occur in cases, both when the lungs were infiltrated superiorly with primary encephaloid cancer, and when they contained secondary nodules of the same kind. (*u*) Failure of weight becomes less valuable as a sign of phthisis, the longer the thirtieth year has been passed. (*v*) The discovery of cardiac disease with marked symptoms deposes against, but does not exclude, the existence of active tuberculization. (*w*) The existence of cancer in any organ is unfavourable to the presence of tuberculous disease; but tubercle and cancer *may* co-exist, even in the same lung.

I would here add, for the sake of beginners, a few cautions in the applications of physical diagnosis in phthisis. Never attempt to give a positive opinion as to the actual state of the lungs, where there has been recent hæmoptysis, or when pleuritic effusion, bronchitis, or pneumonia is present: I of course refer to cases where there may or may not be signs of the first stage; if excavation exist, its signs may be unravelled positively in spite of these complications. Trust very very little, if at all, to the conditions of vocal resonance; accept with great caution the evidence of slight changes in respiration, unless they be corroborated by percussion-changes; place no confidence in jerking respiration (even though local) in a hysterical woman,—nor in harsh respiration limited to the right apex in any woman, nor in very very slight dulness at the right front apex in man or woman. Lastly, never give a confident opinion, in a nicely-balanced case, from a single examination; and make examinations in various postures.

VIII.—A. Experience shows that the *treatment* of the phthisical may, with legitimate confidence, aim at either maintaining a *statu quo* state,—at producing slight local and general improvement, or marked improvement of this kind,—at effecting a total removal of all subjective symptoms, while the physical signs remain partially active,—or at accomplishing total removal of the symptoms, and bringing about a quiescent state of the physical signs, while the general health, weight, and vigour, have improved to such an extent, that the patient shall believe himself totally free from disease, and that the medical observer might be disposed to share his opinion, did not passive physical changes remain. I say treatment may legitimately aim at these ends, because, on the one hand, these ends have been actually obtained; and, on the other, the man has not yet appeared who can point to results more perfect than the best of these, as the positive, direct, and ordinary effect of any known system of medication. This latter clause is not in the least at variance with the well-known fact, that phthisis sometimes spontaneously undergoes permanent suspension of its course.

The results, which I obtained at the Consumption Hospital, might, as formerly shown, be thus set down in general terms, and justify the foregoing statements :—

1. Of a given mass of patients entering the hospital in all stages of the disease, and in every variety of general condition—between the actually moribund state and that of but slight constitutional suffering—the number leaving it, on the one hand, *improved* or *unadvanced* was more than double that, on the other hand, leaving it in *a worse state* or *dying within its walls* (the exact ratio is 67·84 : 32·16). If the cases in which death was actually imminent at the period of admission, were excluded, the result would be very materially more favourable than this. 2. In 4·26 per cent. of the cases, complete restoration to health, not only as regards apparent disturbance of the functions generally, but as regards local evidence of active pulmonary disease, was effected. 3. Complete removal of symptoms was more frequently effected in the male than in the female; but, on the other hand, the results were, on the whole, slightly more favourable in the latter than in the former sex. 4. All patients whose condition grew worse, while they were in the hospital, had reached the stage of excavation on admission; and all patients, whose tubercles were yet unsoftened on admission, left the hospital either improved, or having had a *statu quo* condition kept up. Improvement is more probable than the reverse, even where excavation exists on admission. 5. In a given mass of cases, the chances of favourable influence from sojourn in the hospital will be greater, in a certain (undetermined) ratio, as the duration of the disease previous to admission has been greater,—in other terms, natural tendency to a slow course is a more important element of success in the treatment of the disease, than the fact of that treatment having been undertaken at an early period. 6. The mean length of stay in the hospital in the most favourable class of cases, nearly

doubled that in the least favourable. 7. The chances of benefit are more in favour of those whose trades are pursued out of doors (wholly or partially), than of those who work altogether within doors. 8. The results did not appear to be influenced by the laborious or non-laborious character of the trade individuals might have pursued. 9. The age of the sufferers did not exercise any very material influence on the character of the results. 10. Patients coming from the country have, on an average, a slightly stronger chance of improvement, than the residents of London and the suburbs. 11. Patients admitted during the warmer half of the year, benefit by a sojourn at Brompton, to a slight extent, more than those received during the six colder months.

B. My task in examining the efficacy of various *specific* agents will be brief. Iodide of iron, chloride of sodium, liquor potassæ, chlorine and iodine inhalations, hydrocyanic acid, creasote, digitalis, are disposed of in the masterly analyses of their claims by M. Louis; and naphtha may be allowed to remain in the rather rough grasp of the British and Foreign Medical Review. But cod-liver oil cannot be so lightly dismissed.

I began to employ the oil at the Consumption and University College Hospitals seven years ago, urged to the step by the strong advocacy of Dr. Hughes Bennett, and took an early opportunity of testifying to its remarkable powers in tuberculous and other scrofulous diseases.* The conclusions at which I have arrived concerning its use in phthisis, are as follows. 1. That it more rapidly and effectually induces improvement in the general and local symptoms than any other known agent. 2. That its power of *curing* the disease is undetermined;—I mean here, by "curing" the disease, its power of causing, along with suspension of progress, such change in the organism generally, as shall render the lungs less prone to subsequent outbreak of tubercles, than after suspension occurring under other agencies.†

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* Nature and Treatment of Cancer, p. 202, 1846.

† That such cures really occur in rare instances (and they are as perfect as in any other organic disease, when they do occur) is indubitable. It has been the vanity of late years to deny this absolutely, because a scientific (or pseudo-scientific?) explanation of the fact cannot be found. I am not one of those who refuse to accept the evidence of my senses, because I am unable to com-

3. That the mean amount of permanency of the good effects of the oil is undetermined. 4. That it relatively produces more marked effects in the third, than in the previous stages. Opinions the most diverse have been held on this point; M. Taufflied[*] taught that it had little or no effect on phthisis, if at all advanced; M. Péreyra[†] *reduced the size of cavities in a few weeks* by its administration. 5. That it increases weight in favourable cases with singular speed, and out of all proportion with the actual quantity taken;—that hence it must in some unknown way save waste, and render food more readily assimilable. 6. That it sometimes fails to increase weight. 7. That in the great majority of cases, where it fails to increase weight, it does little good in other ways. 8. That it does not relieve dyspnœa out of proportion with other symptoms. 9. That the effects traceable to the oil in the most favourable cases are: increase of weight, suspension of colliquative sweats, improved appetite, diminished cough and expectoration, cessation of sickness with cough, and gradual disappearance of active physical signs. 10. That in some cases it cannot be taken, either because it disagrees with the stomach, impairing the appetite (without itself obviously nourishing), and causing nausea, or because it produces diarrhœa. 11. That in the former case it may be made palateable by association with a mineral acid; and in the latter prevented from affecting the bowels by combination with astringents. 12. That intra-thoracic inflammations and hæmoptysis are contra-indications to its use, but only temporarily so. I have repeatedly given the oil within a day or two of the cessation of hæmoptysis without any return taking place. 13. Diarrhœa, if depending on chronic peritonitis, or secretive change, or small ulcerations in the ileum,

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prehend what they teach me, and in this matter echo the sentiments of the physician in a recent French tale, speaking of a phthisical recovery:— . . . . "ces miracles de guérison, aux quels la Science ne croit pas, faute de les comprendre, *et devant lesquels je me prosterne, en priant la bonne et sage Nature d'en être moins avare.*"—Pierre, par Madame Reybaud.

[*] Gaz. Méd. de Paris, Nov., 1839.

[†] Du Traitement de la Phthisie. Bordeaux, 1843.

is no contra-indication to the use of the oil; even the profuse diarrhœa caused by extensive ulceration of the large bowel is not made worse by it. 14. That the good effects of the oil are *cæteris paribus* directly as the youth of those using it,—a singular fact, which probably may one day (when the textural peculiarities of youth and age are better understood) aid in giving a clue to its mode of action.

Of the three kinds of oil,—the brown, light brown, and pale,—the brown, I believe, as matter of actual experience, to be the most efficacious.* But, though taken greedily by infants, it is more distasteful than the pale to the adult palate, and hence in grown persons I have been forced to use the latter, less active, kind (in fact, *gild the pill*), in order to ensure oil being swallowed at all. Chemists give no positive answer to the question, on what depends the efficacy of the drug? Its influence on the composition of the blood is yet undetermined. A single analysis by Simon shows a state of hyperinosis, combined with a great excess of albumen, may follow on its use; the solid constituents were in large amount. The patient had been bled repeatedly for hæmoptysis. The iodine of the oil, its phosphorus, butyric acid, gaduine, biliary material, and its mere fatty matter have been severally accorded the chief part in the beneficial results. The discussions on this point do little more than exhibit the existing poverty of our knowledge of the intimate action of remedies. On the other hand, the established efficacy of the oil—a substance of which *à priori* views would scarcely have admitted the possible retention by the phthisical stomach—is another of the conquests of *experimental* therapeutics.†

The dose of the oil at the outset should never exceed (often fall short of) a drachm twice daily: it may be taken in water, milk, orange wine, or any aromatic water agreeable to the patient. The dose may be gradually raised to half an ounce, twice, or at most thrice, in the twenty-four hours. I have never seen any good, and often observed ill, effects follow the attempt to pour in large quantities.

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* On the properties of these varieties of oil, see De Jongh on Cod-liver Oil, by Carey, Lond., 1849.

† As is well known, cod-liver oil has been a household remedy from time immemorial in the north of Europe.

C. The *hygienic* management of consumptive patients is of extreme importance. Attention to the state of the digestive organs, moderate and frequent exercise in the open air, either active (riding and walking), or passive (carriage, swinging, or yachting), the cold, tepid or warm bath, with friction of the skin, the use of flannel next the skin, loose lacing of the stays in females, exercise of the respiratory muscles and lungs by deep inspirations, reading aloud, and movements of the arms, are all of essential service. Exchange of profession or trade, from the sedentary and laborious to the more active and those but slightly taxing the intellect, is advisable. Change of climate, when it can be accomplished, is unquestionably, in the early period of the disease, of fundamental service. Want of faith in its efficacy comes of the change being too often effected when extensive disorganisation of the lungs has already taken place, and is still actively advancing. In the selection of a climate for any particular case, the dry or moist character of the attending bronchitis, and the general tendency to the *strictum* or the *laxum* in the organism, furnish the best guides: in the former case, the climates of Madeira, Teneriffe, the Azores, Rome, Pisa, Torquay, Penzance, Ventnor, and the Undercliff generally; in the latter, those of Egypt, Cadiz, Algiers, Genoa, Nice, Clifton, and Bournemouth, are the most advisable for winter residence. But there are many subsidiary points to be carefully weighed in the choice of winter, spring, and summer residences for the consumptive invalid; and for full information on these I would refer the reader to the classical volume of Sir James Clark.*

. The winter may be passed without danger in-doors in England in an artificial climate, provided proper precautions be taken in regard of ventilation. Air heated to a fixed temperature,* and perpetually renewed by a scientific system of ventilation, such as Dr. Arnott's, forms an excellent substitute for the naturally soft air of milder climates than our own. If, on the one hand, there be the drawback of want of open-air exercise, there are the counterbalancing advantages of the comforts and familiar friends of home and the escape from the labour of travelling.

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* Sanative Influence of Climate, 4th Edit., 1846.

D. The *palliative* treatment of phthisis is a subject so vast, that I can here merely enumerate some of the chief agents that have appeared useful in this regard. Counter-irritation, in the various forms of repeated flying blisters, ammoniated liniments, croton oil (tartarised antimony is the least valuable agent of the class), the strong acetic acid and turpentine, is one of the most important remedies. Special irritability of skin and very great emaciation are the only barriers to the use of these agents, which I prefer as a rule to permanent blisters, issues or setons under the clavicle. The addition of tincture of iodine in variable proportions has appeared to me beneficial; but absolute proof of its promoting absorption I am unprepared to give. Although sceptical as to the professed theory of its action, I have seen benefit derived from liquor potassæ combined with a sedative and bitter tonic. Digitalis and hydrocyanic acid are both useful in cases where the heart is irritable,—checking sometimes, indeed, to a very remarkable degree, various symptoms dependent on that irritability. Where a general state of erethism exists, opium, in some of its forms, must be employed independently of necessity for it as a hypnotic or anodyne. I have never observed results justifying faith in iodide of potassium as an internal remedy; but the syrup of the iodide of iron, especially if there be the least anæmia, is a valuable medicine; it does not increase fever, and sometimes enables a patient to bear cod-liver oil, who had previously failed in the attempt to take this. Mineral acids, with light vegetable bitters, improve the appetite, and control undue action from the skin. Quinine is rarely borne well, and produces no specific effect on the hectic fever.

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* Some latitude may be allowed, of course, for individual taste; but, as I found from questioning upwards of one hundred patients at the Brompton Hospital, an atmosphere at all below 64° Fah. is disagreeable to the majority.

Whatever the theory of Broussais may plead to the contrary, general experience recognises not only the inutility, but the actual mischief, of bleeding, general or local, with the idea of curing consumption. True, intercurrent congestions and inflammations in the thorax may require, both for themselves and to avert their possible consequences, slight abstractions of blood; but it is remarkable how effectually minor attacks of the kind may be counteracted by dry-cupping, blisters, and small doses of antimony.

The treatment of phthisis by daily emetics (supported by a peculiar, but not altogether sound, theory of the site occupied by tubercle) cannot appeal to experience in its favour. Unquestionably it does less mischief, however, than the morbid anatomy of the stomach in this disease would lead us to expect.

I have no experience of dry inhalations, whether of oxygen, hydrogen, or carbonic acid; or of moist inhalations, chloruretted or ioduretted. The latter, doubtless, relieve some forms of phthisical bronchitis. The inhaled vapour of warm water, impregnated with emollient herbs, such as *althæa officinalis*, or narcotic extracts, palliates cough, dryness of throat, &c.

E. The secondary conditions of phthisis often require special treatment. *Hæmoptysis* has already been spoken of. The relief of *cough*, which has resisted opiates and ordinary medicines, may often be effected by the application of three or four leeches above the sternal notch. *Nausea and vomiting* may be controlled by effervescing draughts, Seltzer water, plain iced water, by prussic acid, by a combination (which I have found very useful) of creasote, stramonium and hop; if there be acidity, by liquor calcis or liquor potassæ, and by blisters and sinapisms; or (if there be tenderness) by leeches to the epigastrium. *Diarrhœa* may be arrested by soothing laxatives (rhubarb with magnesia, or castor oil, cajuput, and tincture of opium,) when dependent on irritant matter in the bowel. Sometimes diarrhœa, without evidence of inflammatory action, is more or less amenable to the whole class of astringents,—among which I may specially refer to sulphate of copper and opium, gallic acid and acetate of lead; and enemata of starch and laudanum, or of hæmatoxylon, krameria, or tormentilla. Friction of the abdomen with rubefacients, if there be evidences of sub-inflammatory action, or a few leeches either to the tender part of the abdomen or to the anus, sinapisms, blisters, and emollient poultices, are advisable. The hip-bath is very hazardous. Where the large bowel is extensively ulcerated, remedy after remedy will probably be tried in vain. I have seen some benefit even in these cases from the sulphate of zinc in two grain, and the nitrate of silver in one grain, doses: enemata of the latter, in solution, are also sometimes useful. In all forms of diarrhœa (except when it has become confessedly irremediable) the diet should be low and as dry as possible: thirst may be mitigated by dissolving small pieces of Wenham-lake ice in the mouth. *Perspiration* may be controlled by avoiding much drink and using light bed-clothes; the chest may be rapidly sponged at bed-time with tepid vinegar-and-water, or with decoction of oak-bark. Bark and the mineral acids, gallic acid and lead, have, as medicines, the most perceptible effect. *Inflammatory and ulcerative changes in the larynx* may be stayed by local leeching, blistering, counter-irritation with equal parts of spirits of turpentine and croton oil,* or an issue to the side of the neck or nucha. Some benefit, even temporary improvement in the voice, occasionally follows stimulation of the internal surface of the larynx, with strong solution of nitrate of silver (a drachm to the ounce of water); but I have never seen more than temporary benefit from the process in phthisical laryngitis. *Chronic peritonitis* requires the application of leeches, and blisters (which may be dressed with morphia, when the pain is severe), and friction with ioduretted liniments; emollient poultices with laudanum (if not too thick and heavy) relieve pain. If there be much ascites, diuretics are indicated; but they often fail. In point of fact, this is one of the most uncontrollable conditions of tuberculization; it sometimes undergoes a questionable kind of spontaneous cure. Cod-liver oil, if not already in use, should, of course, be administered: the existence of diarrhœa is not a contra-indication. *Tubercular meningitis* is yet more difficult of cure; moderate local bleeding, purgation, and other revulsive measures, and calomel internally, are alone to be trusted to. I have as yet seen but one even seeming example of recovery in an adult, where the symptoms of tubercular meningitis were positively established. A few prominent particulars of the case will be found in the Appendix. My actual experience on the question whether *fistula in ano* should be cured in phthisical patients, is small; such as it is, it deposes emphatically against interference, unless (what is very rare) there be wasting discharge and serious suffering.

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* This combination must be very cautiously used,—three or four drops only being rubbed in with a piece of flannel for a single minute at a time.

F. The diet of phthisical patients should be nutritious and non-stimulant,—wine and spirits being, in ordinary cases, avoided. I have, however, in special cases of *non-febrile* phthisis, with cold skin, lividity of face, and dyspnœa, prescribed brandy, medicinally, with the good effects of raising the temperature and relieving the dyspnœa. Perhaps the notion that brandy supplies pure aliment for respiration may explain the fact, that habitual spirit-drinkers (*placed otherwise under favourable hygienic influences*) do sometimes suffer less, and live longer, with excavated lungs, than sober persons. I have seen some three or four most remarkable examples of the kind,—examples, indeed, which first led me to a cautious use of brandy medicinally. Gum Arabic, another aliment of respiration, is not open to the objections that attach to alcoholic fluids, and should be allowed largely as an article of food. The mucilaginous material of the Carragheen and Iceland mosses is useful, as satisfying appetite somewhat without exciting the pulse. As respects the quantity of animal food allowable, no general rule can be laid down; so much as each stomach can digest, without local suffering or systemic disturbance, may be safely permitted. Without being an advocate of the "mutton-chop and porter" plan, I am deeply convinced that a low diet is seriously injurious. Light bitter ale, if it do not excite cough, may be permitted in small quantity at dinner. Sarsaparilla agrees with many stomachs, as a diet drink.

Acute Phthisis.

I. When phthisis proves fatal within ten or twelve weeks from the first appearance of symptoms, it may fairly be said to have run an acute course. I have seen, in cases of death under these circumstances, the three following anatomical states: (*a*) limitation of tuberculous deposit, softening and excavation to the apices, just as in ordinary chronic phthisis,—no peculiarity existing in the anatomy of the disease to account for the rapidity of death,—which, however, I have not observed, in this form, earlier than the eleventh week; (*b*) general accumulation of crude tubercles through both lungs, softened irregularly, small excavations existing in various parts, and patches of hepatisation presenting themselves here and there (*acute softening form*); (*c*) general studding of both lungs with semi-transparent grey granulations, coupled with the first stage of pneumonia, that of bright arterial injection, or hepatisation (*acute miliary form*).

Now, in the first case (*a*), there is nothing peculiar in the signs or symptoms; the progress of the disease, except in regard of its rapidity, is exactly the counterpart of that observed in chronic phthisis; and a lurking suspicion frequently remains in the observer's mind, that tubercles may have existed in the latent state, for a greater or less time, before the outbreak of symptoms.

In the second and third cases, there is not only a still greater rapidity of course (death may occur in less than three weeks from the outset), but the signs and symptoms are peculiar, and far from being as significant of the existing disease as might be wished.

II. In the case (*b*) where the lungs are more or less crammed with softening tubercle, the physical signs are as follow:—Inspection and mensuration (in rest) disclose nothing special; application of the hand may detect some increase in vocal vibration, and some deficiency of chest-motion; but it is impossible to say in what part of the chest this deficiency may be most marked. The resonance under percussion is more or less impaired,—at first in some limited points, subsequently over the surface pretty generally; but to the last hour some spots may give resonance not positively abnormal. The respiration, weak in some points, exaggerated, harsh, bronchial, even tubular, in others, is accompanied at first with dry bronchitic, or subcrepitant and mucous rhonchi, the latter of which passes rapidly in various regions of the chest into the thin cavernulous variety with metallic quality. The vocal resonance varies in character.

In the third case (*c*) of acute miliary tuberculization, inspection, mensuration, and application of the hand, give results either completely negative, or those observed in acute bronchitis, —results more calculated, consequently, to mislead than to enlighten the observer. Neither does percussion furnish any trustworthy sign; the conditions of dulness are not only, absolutely speaking, very slight in amount, but they so equally pervade both lungs, that any slight defect of resonance discoverable would naturally be ascribed to individual peculiarity. The respiration is uneven, harsh, slightly bronchial, mingled here and there with dry bronchitic rhonchi, or with sub-crepitation: vocal resonance gives no sign to be trusted to. If pneumonia be present, its signs are of course to be found.

III. The symptoms of acute phthisis in the forms *b* and *c*, are those of a febrile affection, with more or less positive functional implication of the lungs. The invasion, often occurring in a state of apparent health, is marked by rigors, followed by acrid heat of skin; the rigors may occur on several successive days, and there may subsequently be perspirations with sudamina; thirst; anorexia; restlessness; insomnia; prostration of strength; slight lividity of face; dry lips and tongue; wandering, or actual delirium at night; pain in the chest, variable in seat and never intense; cough, either absolutely dry or accompanied with expectoration of clear or yellowish and opalescent mucus, or in rare instances of viscid sputa, slightly stained with blood, without actual hæmoptysis; dyspnœa of considerable amount, indicated not only by the absolute frequency of breathing, but by perversion of its ratio to the circulation; constitute the mass of symptoms ordinarily observed. These symptoms are little influenced by treatment. No abdominal disturbance of any kind necessarily exists; but accidental diarrhœa may occur. The heart and its membranes remain unaffected.

IV. Taking the symptoms now enumerated in connection with the physical signs of each form of acute phthisis, can the diagnosis of either be established? The softening form, *b*, may at first be diagnosticated through the intensity of the general symptoms, contrasting, as they do, strongly with the slight amount of pectoral disturbance,—the ordinary signs of pneumonia, intense bronchitis, and pleurisy, being absent; while general and increasing dulness under percussion, coupled with the signs of breaking up of tissue, eventually render error difficult. I say render error difficult, because double pneumonia running on to suppurative destruction of tissue could alone, besides acute phthisis, produce such physical signs; and had such pneumonia existed, it would have been revealed at first by the signs of its early stage.

The diagnosis of the miliary form (*b*), where grey granulations accumulate sparsely through the lung, with intervening arterial injection of tissue, is greatly more difficult. But the difficulty does not turn on any notable similarity of the disease to the ordinary class of inflammations. Bronchitis, when severe, has abundant subcrepitant rhonchus at both bases, does not cause the dyspnœa observed in the acute granulation-process, and is rapidly accompanied with full muco-purulent expectoration. Pleurisy cannot be supposed to be present, for its physical signs are wanting. And if, for the first two or three days, the perverted ratio of the respiration and pulse might lead to the diagnosis of pneumonia (which, be it observed, exists in a certain state), the lapse of twenty-four hours will prove tha common idiopathic pneumonia does not exist,—for the signs of hepatisation are not an iota more obvious than the previous day.

The real difficulty in my experience (the point has already been touched upon by M. Louis) consists in distinguishing miliary phthisis from *typhoid* fever—fever of the Peyerian species. Dyspnœa, prostration, bronchitic rhonchi, duskiness of face, febrile action, dry skin, adynamic state of the tongue, delirium, and stupor, exist in both affections, and may do so to similar amounts. If pneumonia be present, it affords no help in the diagnosis; for it may be supposed secondary to the typhoid fever. The abdominal symptoms and the peculiar eruption of typhoid fever draw the line, to all appearance, positively,—but only in appearance: for abdominal symptoms may be present in acute phthisis, if the intestine be undergoing acute tuberculisation; and although eruption probably exists in all cases of typhoid fever, it certainly escapes detection (possibly from its slight amount) in a few instances. All these difficulties were well illustrated by a case, of which I give the main facts below.*

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* H. Manning, admitted U. C. H., Aug. 9, 1850 (Case Books, Males, vol. v., p. 166), ætat. 23 (unable to give account of himself, subsequently known from friends to have been taken ill, July 19); prostration, stupor, dingy face, nails livid, skin warm, P. 120, R. 42, tongue dry and cracked, sordes on teeth, *spleen* 1½ hand's breadth high, tenderness in right iliac fossa, abdomen of medium prominence, and generally tender, *diarrhœa* for last two days; no sudamina, no distinct typhoid specks; general percussion-dulness at left back, with diffused blowing respiration; same signs less marked at right back; sputa

V. The *treatment* of acute phthisis is far from being well understood,—the rarity with which the disease is diagnosticated explains this.

The tendency to pneumonia justifies cautious use of the lancet, or, in doubtful cases, the local removal of blood by cupping. Counter-irritation at a certain distance from the chest will certainly be serviceable. Purgation must be avoided, from the danger of exciting the tuberculising process in the intestines. Diaphoretics and sedatives are advisable. The inclination to failure of strength, from the first almost, makes the propriety of exhibiting antimony doubtful; mercury seems a more hopeful medicine. The acute symptoms having been successfully combated, the management of the disease becomes that of its chronic form.

CANCER.

I. Cancer of the lung, most commonly of the encephaloid species, occurs in the forms of secondary nodule, and of primary infiltration, accompanied or not with tuberous formation in either mediastinum about the main (especially the right) bronchus.

II. *Secondary nodules* may be perfectly *latent* in regard of symptoms; and if of small size, they are with difficulty detected by percussion, and may not produce pressure enough on the lung-substance to modify the respiratory sounds. Bronchitis occurs sometimes; but may be totally absent even where the

viscid, somewhat transparent, of faint tobacco-juice tint; heart's size and sounds natural. *Diagnosis: Typhoid fever, with secondary pneumonia* (the difficulty about the want of eruption being noticed at the time). *Death,* Aug. 14, (the 28th day). There was double hepatisation mainly of the lower lobes; and the entire of both lungs were profusely studded with semi-transparent grey granulations (some growing opaque in the centre); a stratum of recent lymph in the left pleura was similarly studded; the spleen, 6 in. high, weighed 11 oz.; liver fatty; Peyer's patches contained crude yellow tubercles, here and there, size of pins' heads. The obscurity arising from tuberculisation of the intestine has not been referred to, so far as I know, by any author.

nodules are sufficiently close and bulky to cause marked percussion-dulness. Dyspnœa is probably the only symptom distinctly traceable to these nodules.

III. The physical signs of *primary infiltrated* cancer of the lung are numerous. The affected side is flattened, or even generally retracted, and the intercostal spaces slightly deepened; the respiration play impaired; the vocal fremitus increased, if the infiltration be slight; diminished, or even annulled, if it be extensive, or, especially, if coupled with tumor; the heart's impulse may be felt, too, distinctly through the lung. The percussion-sound is dull, or hard, wooden, and even tubular, especially about the third rib; the dulness sometimes extends across the middle line. The respiration is of diffused blowing type, unless the main bronchus undergoes accidental closure from the pressure of a surrounding cancerous mass, when the respiration grows weaker and weaker, and may eventually be almost suppressed. Bronchophony sometimes exists markedly. Should softening and elimination of the infiltrated texture occur, cavernous respiration and rhonchus supervene, unless there be accidental closure by pressure of the main bronchus from superadded tumor. The diaphragm may be raised slightly on the affected side; the non-cancerous lung (for the disease is limited, in the great majority of cases, to one organ) gives signs significant of health, of hypertrophy, of emphysema, or of bronchitis.

Here we have the signs of a disease diminishing the bulk of the lung, and causing retraction of the side. But this disease may be associated with actual tumor (cancerous) in the mediastinum, which, as will hereafter appear, has a dilating influence on the chest-walls. A combination of the signs of the two opposing states is met with in instances of their association.

IV. Primary cancer of the lung may (but this is infinitely rare) run a *latent* course. In the great majority of cases, manifest *symptoms* attend its progress. Pain, of variable duration, intensity, extent, and constancy, apparently occurring either in the cancerous lung itself, in the pleura, or in the intercostal nerves, is a very constant, and sometimes has proved the first, symptom. Dyspnœa, either slight, or troublesome, rarely acquires any great intensity, unless there be co-existent tumor. Cough, an invariable attendant, is, in very rare instances, dry; in the great majority, attended with expectoration. The sputa may be simply catarrhal, purulent, or bloody. In the latter case, the blood seems thoroughly mixed with serosity, mucus or mucopus, and the sputa, commonly opaque, sometimes slightly translucent, are of the colour of red or black currant jelly (and not very unlike those substances), or pink. Encephaloid detritus has in rare instances been expectorated, of cognisable physical characters. The frequency of hæmoptysis has already (p. 349) been referred to. Excessive fœtor of the breath or sputa, with or without positive local gangrene of the lung, occasionally occurs.

Infiltrated cancer produces no eccentric pressure-signs, and the only concentric sign of the kind which it appears to have positively produced, unassisted by tumor, is dysphagia. If associated with mediastinal tumour, the various pressure-signs of that disease will be more or less prominently present.

The general symptoms are sometimes slight. Emaciation is slow in appearing; once established, it advances rapidly. Night perspirations sometimes occur profusely; febrile action is not often highly marked; the pulse varies in characters. The skin commonly exhibits some shade of the straw or waxy cancerous tint. Increasing dyspnœa and cough, insomnia, failure of assimilative power, sometimes attended with anasarca of the lower extremities, (but this rarely occurs to any extent unless tumor be present,) put a term to existence.

V. Infiltrated cancer is distinguished from *chronic pneumonia* by the amount of flattening of the side; by the occasional extension of dulness beyond the median line (which never, so far as I know, occurs in *chronic* pneumonia); by any sign of concentric pressure present (as dysphagia or weak respiration from bronchial pressure), for such never occur in pneumonia; by the progress of the disease, which, stationary in pneumonia, leads to excavation in cancer; by the peculiar jelly-like or cancerous expectoration in the latter disease, and by the much greater severity of its local symptoms. From *tubercle* cancer differs by the signs of extensive consolidation being unattended with rhonchus; the absolute limitation of the disease to one lung (common in cancer, infinitely rare in tubercle); by any signs of concentric pressure, as gradual suppression of respiration on the affected side; the jelly-like expectoration; the less severity of the constitutional symptoms, and the total absence of the secondary morbid states of phthisis. If the cancer have softened, the microscopical characters of that product may be found sometimes in the sputa. From *chronic pleurisy, with retraction,* cancer will be distinguished by the less amount of deepening and narrowing of the intercostal spaces; the greater respiratory play; the less irregularity of surface; the greater amount of respiratory murmur, especially in the inferior regions; the natural positon of the shoulder, scapula, and spine; the absence of friction-sound; by any concentric pressure-sign that may be present; the peculiar expectoration; by the greater severity of the local symptoms as a whole; and the history of the case, indicating in the one instance a disease on the increase, in the other on the decline. If *dilatation of the bronchi and "cirrhosis"* co-exist with chronic pleurisy, and if the bronchi be the seat of active secretion, while the constitution becomes implicated, the case will be less easily distinguished from cancer. But many of the points already enumerated will still avail; hæmoptysis, or the peculiar expectoration will probably have occurred, if the case be cancerous. The existence of external cancer may aid in the diagnosis in any of the above cases.

VI. Cancer seated in the lungs is even less under the control of remedies than when occupying some other localities. No evidence exists of the utility of arsenic or conium; cod-liver oil deserves a trial. Symptoms must be relieved as they occur, by ordinary measures. Dyspnœa is best mitigated by dry-cupping and blistering; small bleedings give temporary relief, but they cannot, of course, be often repeated.

DISEASES OF COMPOUND CHARACTER.

SPASMODIC ASTHMA.

I. By spasmodic asthma is understood paroxysmal dyspnœa, of nervous (and reflex) origin, solely or mainly; and existing either independently of any organic change in the lungs, or, as is much more common, connected with emphysema.

II. A paroxysm of asthma is either preceded by various disturbances (digestive or other), or occurs suddenly, often in the night, without the slightest warning. The patient starts out of sleep, with a feeling of suffocation or constriction about the chest; the efforts at inspiration, convulsively violent, are accompanied with sinking of the epigastrium, and elevation of the diaphragm (evidently from the diminished mass of air in the lungs); the expiration is prolonged and comparatively easy; both acts, but chiefly the former, are attended with wheezing. Various postures are assumed to facilitate the attempt at filling the chest,—the patient stands erect, or leans the head forwards between the hands, or places himself on his knees and elbows,—or rushes to an open window, and gasps wildly for air. The pulse is small and feeble, sometimes irregular, whether there be or be not palpitation of the heart; the eyes prominent and staring, the face flushed, livid or pale, the extremities cold, the skin clammy, the look anxious and imploring. The urine, pale and abundant during the paroxysm, becomes scanty and high-coloured at its close. The fit may last from two to four hours, terminating by expectoration or not (humid and dry varieties), and leaving, on its cessation, a sensation of fatigue and prostration.

The physical signs are rather negatively than positively valuable. There is much laboured and jerking elevation-movement of the thorax; little or no true expansion. The gradual emptying of the lung of its residual air, which takes place in the paroxysm, slightly impairs the resonance under percussion.* True inspiratory sound may be almost completely deficient, sibilant or sonorous rhonchus taking its place. Laennec pointed out that if the patient be directed to speak, without drawing breath, as long as he possibly can, so as to exhaust the chest as completely as possible, the next two or three quiet inspirations produce well-marked murmur;—temporary relaxation of spasm, probably, has permitted the air to reach the vesicles.

Repetition of asthmatic fits may lead to dilatation of the right heart, and insufficiency (without organic disease) of the tricuspid valve; but this is very rare, unless there be emphysema, as a permanent malady.

III. The *treatment* of spasmodic asthma embraces that of the fits, and of the constitutional tendency to their recurrence.

In the fit, our effort must be to relax spasm. In a first attack, if there be great congestion, bleeding may be advisable; but in the subject of habitual seizures, it is altogether inadmissible. Nauseating expectorants, ipecacuanha, lobelia, squill, or colchicum (if the patient be gouty), or an emetic of sulphate of zinc, sometimes cut short a fit very obviously: various antispasmodics, sulphuric æther, asafœtida, musk, and very strong coffee mitigate the intensity of the paroxysm. Of narcotics, stramonium, cannabis indica, belladonna, and opium, are the most effectual. The fumes of burning paper, saturated with nitre, sometimes shorten a paroxysm, by inducing copious expectoration; but they oftener fail. Electro-galvanism; the cold water dash over the shoulders; draughts of cold water, while the feet are immersed in a mustard foot-bath,—are all measures that occasionally relieve. The effects of chloroform inhalation are variable; I have seen three kinds of result: total relaxation of the spasm during the continuance of insensibility, with immediate return of dyspnœa on restoration of consciousness,—gradual return of the difficult breathing, after consciousness is restored,—and suspension, or at least mitigation, of the paroxysms for the time being. The last effect is the rarest of the three; but, on the other hand, the temporary relief afforded by chloroform is *sometimes* more complete and more rapid than by any other agent that I know of.

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* This I repeatedly ascertained in a girl, named Harmer, a patient both of the Consumption and U. C. Hospitals; but, on the whole, this sign is rarely to be established.

A coming fit may sometimes be prevented by a cigarette of stramonium and belladonna, by strong coffee, by mental excitement or amusement, by heating the body generally to as high a degree as can be borne, &c.

The tendency to paroxysms may sometimes be removed by counter-irritants to the dorsal spine, or to the nucha, by strychnia in very minute doses, by electro-galvanism steadily repeated in the course of the vagi and phrenic nerves, by great attention to the state of the alimentary canal, caution in eating (both as to quantity and quality), and the use of certain metallic tonics, especially the nitrate and oxide of silver, and the sulphate of zinc. Change of air is most important; but the kind of change that shall prove beneficial can only be learned by experience. Some sufferers lose their paroxysms south of the olive line, others are easiest in a cold atmosphere; moisture, the bane of some, greatly mitigates the disease in others. The air of towns suits some, that of the country others; the clear suburban air of London is infinitely more noxious to some asthmatic persons than the foul atmosphere of the worst cleansed and most densely peopled localities of the metropolis;* occasionally an individual will be found who is tortured with asthma in one room of a house, free from it in others,—and this without any distinct explanation being found in the aspect, the drainage, or any other known condition.

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* A man, one of the greatest sufferers from asthma I ever saw, lived in the neighbourhood of Chalk Farm, the pure air of Hampstead blowing across his house. I tried, I believe, almost every known remedy, in vain, for his relief. He was accidentally detained one night in the foul region of the Seven Dials; feeling persuaded he could not possibly survive till morning, so great was his dread of the close air. He not only lived through the night, however, but enjoyed the first uninterrupted sleep he had known for months. He took the

PARALYTIC ASTHMA.

If there be motive to believe that nervous asthma commonly depends on spasmodic (reflex) action of the bronchial muscular apparatus, there are many speculative reasons (for instance, the comparative facility of inspiration, and difficulty of expiration, noticeable in some asthmatics) for presuming that paralysis of the apparatus may cause a variety of the affection. Possibly such cases are those benefited by strychnia and galvanism. But demonstration is wanting.

HOOPING-COUGH.

I. Hooping-cough, or pertussis, seems practically composed of reflex spasm of the air-tubes, and special bronchitic irritation, the speciality of character being indicated by the nature of the secreted product.

II. Three stages of the disease are generally recognised: the catarrhal; the spasmodic; the terminal.

The catarrhal stage, lasting from one to two weeks, is marked by coryza, general irritation of the air-passages, dry, or almost dry, and slightly paroxysmal, cough, and feverish action. If there be expectoration, it is in nowise specific in character.

The commencement of the spasmodic stage is announced by the distinctive, abrupt, paroxysms of cough, occurring at

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hint; removed to the Seven Dials for *the benefit of the air;* and when I last saw him, some six months after the removal, continued, though still a wheezer, perfectly free from serious dyspnœa.

irregular intervals by day and night; during these fits both inspiration and expiration are, in their several ways, laborious,—the former prolonged, and accompanied with a loud, cooing noise, called hooping (obviously caused by partial closure of the glottis); the latter consisting of a number of successive forcible puffs, without obvious intervening inspiration, performed with almost convulsive energy. These successive expiratory efforts seem to force almost all the residual air from the lungs, and it is often not till some of the minor phenomena of asphyxia become apparent, that the patient gets momentary relief, by the prolonged cooing inspiration. A paroxysm consisting of a variable number of these inspiratory and expiratory efforts may last from less than a minute to a quarter of an hour or upwards; when thus prolonged, not only do the face and eyes become painfully turgid and livid, but blood may issue from the mouth, ears, and nose; the conjunctiva become ecchymosed; slight convulsions occur, and involuntary (probably expulsive) discharge of the fæces and urine take place. The pulse is frequent, greatly out of proportion to the number of inspirations; but it becomes natural (unless there be complication present) after the close of the fit. The paroxysm terminates commonly by expectoration; during the early part of this stage, thin, scanty, and pituitous; during its more advanced part, ropy, semi-transparent, albumen-like, or by vomiting,—or by pure exhaustion, without discharge of any kind. And in a few minutes, sometimes a few seconds, the patient feels as in health, except some slight sensation of fatigue, and, if a child, resumes his play, as though nothing had happened. The frequency of recurrence of the paroxysms varies greatly; there may be but two in a day, or two or three per hour. Sometimes ensuing without any immediate excitation, they are more frequently traceable to such influences as irritate the excito-motory system,—the act of swallowing, sudden draughts of cold air, a fit of anger, abrupt movements of the body; like spasms of reflex mechanism generally, they often come on by night. The ordinary duration of this stage ranges from six to eight weeks; but it may terminate by the third week, or continue for months.

During the terminal stage, the cough becomes less frequent, loses the convulsive and hooping characters, the expectoration ceases to wear the albumen-like, ropy, appearance, and becomes simply catarrhal. Eventually all symptoms disappear in the course of from one to three weeks.

Hooping-cough is in some cases an affection so mild, that doubts may be entertained as to its having existed at all; or so severe as to destroy life. Death has occurred from asphyxia, from convulsions, from rupture of the air-cells, and effusion of air into the mediastinum and common cellular membrane, or from rupture of the pleura and pneumothorax, or from the mere exhaustion of the protracted disease. Various of its complications, bronchitis, pneumonia, croup, or convulsions, also occasionally prove fatal.

III. The physical signs are only negatively important: they exclude diseases which might erroneously be supposed to be present. During prolonged paroxysms, and especially towards their close, the percussion-sound falls somewhat in clearness: I state this from positive observation; but it is no more than might be expected from the forced evacuation of the air-cells that occurs. During the brief efforts at inspiration between the successive expiratory puffs of the cough, inspiratory murmur may sometimes be caught; but during the noisy and hooping inspiration, very little true breathing-sound reaches the surface, —probably spasm of the bronchial tubes prevents the air from advancing to the vesicles. Sonorous and sibilant rhonchi, and, if there be much fluid in the tubes, the varieties of the moist bronchial rhonchi, are heard.

IV. In mild cases of hooping-cough little is required in the way of *treatment*. Attention to the state of the bowels, limitation in eating, warm clothing, and avoidance of all the *lædantia*, is all that is called for.

In some cases during the catarrhal stage, purgatives and antimony, or ipecacuanha, in doses measured by the patient's age, are desirable. Unless the symptoms are sharply inflammatory, blood-letting in any of its forms should be avoided; the disease must run a certain course, and the patient's strength requires husbanding.

Various means of lessening the severity of the paroxysms may be had recourse to. Nauseants, given also occasionally in emetic doses, are among the best of these,—antimony, ipecacuanha, and lobelia inflata, especially the two latter, in combination. Antispasmodics, such as asafœtida (if unbearable by the stomach, rubbed over the epigastrium and spine), musk, valerian, cochineal, and camphor, answer well in some cases. Of narcotics, hyoscyamus, lettuce, and conium are the safest; opium should not be given unless in combination with ipecacuanha. Belladonna, pushed to the verge of poisonous effects, is sometimes a justifiable remedy, where the paroxysms are dangerous *per se*; but I have no evidence that it shortens the disease. Hydrocyanic acid, as a rule, is a fitter agent. Tonic medicines, oxide of zinc, nitrate of silver, and iron, are sometimes required before the close of the disease.

Counter-irritation by tartar emetic, croton oil, flying blisters, &c., is decidedly useful: liniments should be used of a mild kind. Belladonna frictions to the spine, or a broad strip of belladonna plaster extended from the nape of the neck to the loins, are distinctly serviceable in many cases. Morphia, applied endermically to the throat, is favourably spoken of. A fit may sometimes be shortened by a draught of cold water, or by the cold water dash to the face. The shower-bath, in the case of children, generally does more harm than good.

In infants, the teeth should always be carefully looked after; flannel should be worn next the skin, a uniform temperature maintained about the patient as far as possible, and, if the complaint set in towards winter, and removal to a warm locality be impossible, he should be confined to the house absolutely.

Towards the close of the disease, change of air (just as in laryngismus stridulus) proves of striking benefit.

Of various alleged specifics, such as sulphuret of potassium, nux vomica, arsenic, and cantharides, the less said the better.

INTRA-THORACIC TUMOR.

I. I have seen in the mediastina tumors, composed of simple exudation-matter, scirrho-encephaloid, encephaloid, fibrous, and fibro-fatty substance: the local symptoms and the physical signs (inasmuch as both are mainly of mechanical origin) are almost identical, whatever be the constitution of the tumor.

II. When a tumor occupies the mediastina, and encroaches, as it commonly does, mainly in front, bulging of the sternum and costal cartilages, in a variable spot and to a variable superficial extent, may exist; the intercostal spaces, widened and flattened, are then unaffected by respiration; but, on the other hand, no shadow of bulging may exist, and yet from other signs the presence of tumor be indisputable. Wherever a tumor, of any thickness, reaches the surface, vocal fremitus is annulled; fluctuation, simple or peripheric, is not to be detected; and a double impulse is sometimes transmitted from the heart, and may be felt both by observer and patient as a sort of inward succussion. If the tumor encroach pretty equally on both sides of the chest, there may be no alteration in their relative semicircular measurement; the respiration-play is found to be impaired, and the impairment may fall rather on expiratory retraction than inspiratory expansion. If by chance either main bronchus (and it is much more frequently the right that suffers) be seriously obstructed, the respiration-play will be relatively deficient on that side, independently of the influence of any excess of tumor within the right thorax. Should the tumor encroach notably on one side or the other, and be at the same time adherent to the wall of the chest, the space between the middle line and nipple will be lengthened on the same side. Wherever tumor reaches, or closely approaches, the surface, percussion will furnish an excessively dull short sound, with highly marked parietal resistance; the superficial dimensions of the growth may thus be traced in the front or in the spinal regions behind. The resistance and resonance of the heart and liver commonly differ from those of tumors; and hence, if the natural and morbid structures are in juxtaposition, their neighbouring edges may be defined by simple or by auscultatory percussion. Near the trachea, or more generally in front, or even in the back, the percussion note may be tubular, or even amphoric. I have known it amphoric over the lower half of the right back. The auscultatory signs vary widely. The respiration may be weak, almost to suppression over the morbid mass, or (from pressure on the main bronchus) over the side generally; or it may be of diffused or even tubular or hollow blowing type: these differences will depend on the precise relationships of the growth to the bronchial tubes and parietes; as will the absence or presence of bronchophony or pectoriloquy of the loud form. The heart's sounds are conveyed through the solid mass with undue intensity, and if this press on the aorta or pulmonary artery, there may be systolic murmur, simulating intra-cardiac murmur at the base. Bronchial dry and moist rhonchi may be heard. The position of the heart and of the wings, or of either wing, of the diaphragm, will depend wholly on the direction in which the tumor grows: they may be considerably displaced, or retain their natural positions, even where a large mass occupies the mediastinum.

Certain symptoms arise, or may arise, mechanically from pressure of the growth. *Eccentric* pressure causes bulging of the thoracic parietes, as we have seen, and depression of the diaphragm. *Concentric* pressure may produce dysphagia, sometimes, though rarely, of excessive pertinacity, by acting on the œsophagus; playing on the superior cava or innominate veins, it causes distension of the jugular, subclavian, axillary, mammary and superior epigastric, facial, frontal, and even dorsal veins, coupled with a tumid spongy fulness of the base of the neck, swollen livid discoloration of the face and lips (which look in extreme cases distended almost to bursting), and œdema of face, arm, and affected side of the thorax or the chest generally; encroaching on the inferior cava, pressure entails anasarca of the lower extremities; influencing the innominate or either subclavian artery, it weakens either radial pulse; forcing backwards, or to either side, the trachea, it induces stridulous breathing and weakness of voice (traction of the recurrent nerve may have something to do with both these symptoms); its effects on the main bronchus have already been referred to, and sequential to these, exaggerated respiration may be established in the other lung. Irritation and pressure-action combined may give rise to active hydro-thorax or actual pleurisy with effusion.

III. The symptoms of intra-thoracic tumor are pain, very variable in amount; dyspnœa; cough, with or without expectoration, of the jelly-like kinds (p. 412), or catarrhal; hæmoptysis; inability to lie with the head low, and eventually complete orthopnœa, the patient sometimes, for weeks before death, never daring to go to bed, and never enjoying more than fitful dozes. The constitution may long bear up against the local disease; but the dyspnœa and insomnia at length affect the appetite, and emaciation sets in. The patient dies gradually, anasarcous and exhausted, or he may perish suddenly from obstruction of the pulmonary artery.

IV. An intra-thoracic tumor may, according to its seat, be confounded with extensive chronic pneumonia and chronic pleuritic effusion, with chronic pericardial effusion, great enlargement of the heart, or aneurism of the thoracic aorta. The distinctive marks of the cardiac and arterial affections will be found in the descriptions of these.

Tumor will be distinguished from chronic pneumonia by the tendency to increase, instead of diminution, of bulk of the affected side, by the implication of the mediastinum, by the greater intensity of the dulness under percussion, the failure or the disappearance of vocal fremitus, which remains in chronic pneumonia, and the different characters of the respiration in the two diseases. Hæmoptysis and red jelly-like expectoration never occur in chronic pneumonia; whereas the emaciation is of earlier appearance, and more marked than in cases of tumor.

From empyema, tumor will be distinguished by the absence of intercostal fluctuation, simple or parietal; by the greater intensity of dulness under percussion; by the limits of the dulness not being changed by altering the patient's position (p. 284); by the interspaces not being convex; occasionally by the intensity of blowing respiration; by the clear transmission of the heart's sounds; by the comparative irregularity of outline of the side, some spots being more prominent than others; by the fact, that in cases of tumor, careful percussion will almost surely detect some spot giving a comparatively clear sound, where, according to the laws of physics, liquid, had this been the cause of the general dulness, must have made its way, and hence caused dulness there as well as elsewhere; by the whole class of concentric pressure-signs; and, if they have occurred, by hæmoptysis, or by the jelly-like expectoration.

A patient having mediastinal tumor may also have empyema on the side most encroached on, generally the right. If the patient be seen for the first time, when the two diseases are present, and if precise medical information as to the previous course of the complaint be wanting, this combination may be very difficult of diagnosis. But empyema does not produce concentric pressure-signs, nor give rise to hæmoptysis or jelly-like expectoration; if these symptoms be present, there must be, in addition to empyema, either tumor or aneurism. Besides the test of moveableness of dull sound (p. 284), may be appealed to with considerable confidence.

Given an intra-thoracic tumor, how may its *nature* be determined? If the signs of infiltration of the lung co-exist, the tumor, as far as I have seen, is either composed of simple exudation-matter (of these I have examined three microscopically) or cancer. If the tumor present externally, it is cancer; if tumors exist elsewhere, either secondary to, or independent of, that in the chest, supposing even *their* nature cannot be established directly, the chances are strong, that the thoracic growth is cancerous. Violent hæmoptysis is more common with cancerous than other growths; expectoration of cancer can, of course, only occur with the former. The constitutional characters of cancerous disease may be wanting.

V. Intra-thoracic tumor is, of course, beyond the *permanent* influence of *treatment.* But it is astonishing what marked *temporary* improvement of all the direct symptoms may be effected by cautious cupping, dry-cupping, flying blisters, profuse inunction with an ioduretted liniment, gentle purgation, and diuretics. I have twice, within the last year, known the diagnosis of intra-thoracic tumor contested, on the grounds of the relief produced by such measures; yet, *post-mortem* examinations, in one instance in three, in the other, in seven weeks, proved the existence of massy growths. Unfortunately, all therapeutical means soon cease to avail us; and I know no more truly painful spectacle than that of the closing sufferings inflicted by mediastinal tumors.

CHAPTER II.

DISEASES OF THE HEART.

FUNCTIONAL DISEASES.

I. FUNCTIONAL disturbance of the heart is indicated by increased action (*palpitation*), perverted action (*fluttering, or irregularity of rhythm*), and decreased action (*syncope*). These conditions, variously combined, may attend various textural changes in the organ; but occasionally exist, independently of any such appreciable change.

II. The distinction of functional from organic disturbance of the heart is often far from easy. Many of the general rules given for this purpose fail clinically. The inconstancy of functional, and constancy of the symptoms of organic ailment, are strongly dwelt on, for example; but all the subjective, and many of the objective symptoms may disappear temporarily in cases even of extensive organic diseases. The existence of secondary changes, such as subcutaneous œdema, congestion of the lung, &c., commonly proves the cardiac affection to be organic; but not always, for spanæmia, added to nervous palpitation, may induce œdema. If exercise relieves a disturbed heart, its affection is pronounced to be dynamic only; but, if spanæmia exist, exercise may be unbearable. If, in the intervals of attacks of disturbed action, the force and rhythm of the pulse and heart are natural, those attacks are said of necessity to be functional:

an error; for the most perfect tranquillity of the organ *may* exist, from time to time, though its texture is seriously unsound. Careful and repeated physical examination alone will, in difficult cases, justify a positive opinion on this question. And the student must remember that the mere detection of a morbid physical sign in a palpitating heart, does not justify the assumption of organic disease: a basic systolic murmur may be simply anæmic; a systolic murmur at the mitral apex may be generated by irregular action of the musculi papillares; extended dulness to the right of the sternum may depend on temporary distension of the right cavities with blood.

III. In dynamic palpitation, action may be increased very triflingly or sufficiently to shake the entire trunk; the frequency of the systoles, commonly increased, is sometimes greatly so; their rhythm may be regular or irregular.

The impulse is too extensively visible, but the apex-beat natural in site. If the heart be a well-nourished one, the impression it gives to the hand laid on the cardiac region, is that of a *blow* (the impulse may even be somewhat heaving); if a feeble organ, the impression is that of a *slap*. Habitually, the area of dulness remains unaltered, but extension to the right of the sternum may occur, in prolonged paroxysms especially; dulness is never carried upwards. The first sound is too loud and clear at the mitral apex, and somewhat abrupt and short; the second is duller and less clicking than natural at the mid-sternal base. The first sound may be loud enough to be audible, both to the patient and to bystanders, at a distance of some inches from the chest. Reduplication of the second sound at the base is common; and a clear metallic ring, or a pericardial rub, may accompany the shock at the mitral apex.

Will palpitation produce murmur? If there be the slightest co-existent spanæmia, basic systolic murmur will, of course, be generated; but this murmur may also occur, both in males and females, during violent palpitation, where no evidence exists of any morbid state of the blood. I suspect that mere palpitation may also cause passing mitral regurgitant murmur of dynamic mechanism (p. 220); but of this I am not sure.

The aorta, the carotids, and the arteries generally, beat with undue force, sometimes exceeding that of the heart itself; the pulse is quick and sudden, or full, hammering, and heavy, in some plethoric people, whose hearts are free from hypertrophy.

Among the subjective symptoms may be enumerated choking sensations; a feel as if the heart were jumping into the throat; præcordial anxiety, with faintness or actual syncope and insensibility; præcordial pain, slight, dull, aching soreness, or agonising pangs, simulating angina, and sometimes relievable by pressure; hurry of respiration, sometimes out of proportion with the pulse, giving the patient the appearance of a person out of breath with running; tinnitus aurium; vertigo and confused vision; cephalalgia; heat of head and flushed face; clammy coldness of the extremities. In severe attacks there is often extreme general distress, and fear of death,—singularly enough, more of this, often, than in cases of palpitation of organic origin. Lasting for a few minutes, an hour, or with remissions for days together, a fit of palpitation frequently terminates by sleep.

In the *treatment* of the paroxysm, the first effort must be to remove, or lessen the intensity, if possible, of its cause. The fit may be shortened by antispasmodics, asafœtida, musk, valerian (especially in hysterical persons); by diffusible stimulants, ammonia, the æthers, and very strong coffee; by narcotics and sedatives, opium, hyoscyamus, hydrocyanic acid, and if the excitement of the organ be great, and its action not distinctly irregular, by digitalis. Acidity and flatulence, frequent causes of palpitation, may be corrected by soda and cajuput oil; a loaded stomach freed by an emetic. If plethora of the sthenic kind be present, cautious venesection is advisable: if of the asthenic, digitalis tranquillises the organ speedily. If a gouty or rheumatic state be discoverable, colchicum, guaiacum, and ammonia, and irritant applications to the joints, are the best remedies. Heat may be applied to the extremities; but the application of ice over the heart is a dangerous practice, especially if the rhythm of the organ be affected.

Persons subject to palpitation should avoid stimulants, over-exercise, over-sleep, emotional and intellectual excitement. Hydrocyanic acid, aconite, digitalis, and belladonna, varied according to circumstances (the latter also in the form of plaster over the heart), coupled with the use of the shower-bath, attention to diet, regularity of bowels, cheerful occupation, and, lastly, change of air, will either remove the tendency to palpitation altogether, or greatly mitigate the severity of its seizures. If there be the least spanæmia, iron is indispensable.

IV. Perverted or irregular action affects rhythm mainly, force secondarily. The varieties of perversion, as indicated by the heart's impulse, by its sounds, and by the relationship of these sounds to the arterial pulses, have already been described (pp. 172 and 206).

A heart beating, now fifty, now one hundred and eighty or more times in a minute, now with excessive force, the next moment with such feebleness that the hand scarcely catches the impulse, cannot fail to become more or less clogged and obstructed internally. The evidence of this appears not only in the syncopal and suffocative tendencies of such fits, when at all prolonged, but in the increased area of the heart's dulness,—especially to the right of the sternum. The quality of the heart's sounds changes from moment to moment,—the general tendency of the first of the two, especially, is to shortness and undue clearness; but occasional forcible contractions may give a healthy systolic sound, masked possibly by parietal shock.

In this form of palpitation, præcordial anxiety generally reaches its maximum. The prognosis is more serious than in the previous form. Although irregularity of action may exist in the very highest degree without valvular disease, I confess I have very rarely met with it under such circumstances, where there was not either certainty or strong suspicion of alteration of the heart's texture,—in the form of softening or fatty degeneration. At all events, I am persuaded that when elderly persons are the frequent subjects of what is called "stomach-palpitation," the assumption that, because no valvular signs are discoverable, the heart is sound, is a positive error. Excessive irregularity of the heart's action must always be looked on as a serious affair, though exceptional individuals exist, who, by some peculiar idiosyncracy, enjoy good health, with an habitually more or less irregular pulse: in these persons, it is to be remembered, too, the organ is quiet in action, though morbid in rhythm.

During a fit of this form of palpitation, the stimulant and anti-spasmodic plan of treatment is required. In the intervals the tone of the heart may be improved by ferruginous and other tonics, the use of the shower-bath, and removal to a bracing climate.

V. Decreased action, affecting force or frequency, or both, if carried to extremes, produces syncope; and in its minor degrees entails general languor of all the functions.

(*a.*) Actual syncope, whether induced directly by failure of the heart's irritability, or indirectly through failure or perversion of nervous influence, or by loss of blood, occurs with very much the same train of symptoms.

Commonly some premonitory symptoms are noticed: nausea, sinking feel at the epigastrium, disturbed vision, vertigo, tinnitus aurium, confusion of thought, pallor, drawing of the features, inclination to clammy perspiration, tremulous contractions of the muscles, or slight convulsions, chattering of the teeth, and failure of the pulse, announce the coming syncope. In less usual cases the actual stoppage of the heart's action is sudden.

In the state of complete syncope the pulse, though absolutely wanting at the wrist, may often be faintly felt in the carotids; the patient is totally unconscious; the surface cool, clammy, or not; the features contracted; the nares pinched; the lips of marble pallor; the face and skin generally more or less blanched; the respiration suspended absolutely, or almost imperceptible: in some few instances the sphincters of the bladder and rectum relax. The heart's impulse may be almost or completely lost to the eye and hand; the sounds are rarely totally inaudible in ordinary syncope; both are, of course, exceedingly feeble, the first very short, the second generally lost at the apex.

Lasting for an instant only, for seconds or minutes, ordinary syncope terminates by gasping or rather sighing respirations, at long intervals, and gradual return of pulse, consciousness, and colour. Sometimes vomiting or discharge of flatus, convulsions, palpitation, or profuse perspiration, take place at the time of returning consciousness. The sensations are, on the whole, painful and distressing.

Syncopal unconsciousness will be distinguished from that of asphyxia by the pallor of surface; in the latter, congestion and general lividity of the head and face exist.—In uræmic coma, or semi-coma, there is pallor of face to deceive; but the state of the heart's sounds and the fulness of the pulse at the wrist will prevent error.—Apoplexy, though traced *post-mortem* to cerebral hæmorrhage, sometimes occurs with pallor of face; the state of the pulse and of the heart prevents the possibility of error,—in apoplexy, neither pulse nor heart fails in strength, and both may act with undue energy.—Hysterical insensibility, with absolute motionlessness, closure of the eyes, almost complete suppression of respiration, simulates syncope; but there is no pallor, and the pulse beats steadily and with good force. —Lastly, cases of prolonged syncope occur where the pulse is imperceptible, respiration suspended, consciousness gone; and this state continues for several days. Such a condition might easily be, and has been, mistaken for actual death: in prolonged syncope, it is said the countenance retains some life-like expression, and the thermometer marks a higher degree in the rectum and mouth than on the surface generally; besides, neither cadaveric congestions nor rigidity make their appearance.

In doubtful cases sufficient time should be allowed for putrefactive changes, lest a catastrophe, the most horrible the mind can conceive, occur. Never having observed a case of such apparent death, I know not whether the heart's sounds are absolutely annulled: Hope imagines the second might be heard, although the first were inaudible.

In treating an attack of syncope, the first points are to place the patient horizontally with the head on, or below, the level of the shoulders, to allow a free circulation of cool air, and remove all pressure from the neck and chest. If the syncope be caused by loss of blood, a tourniquet may be applied with advantage to one or both femoral arteries. Stimulant impressions on the nerves,—on those of the nostrils and lungs by ammonia, strong acetic acid, the fumes of burning feathers,—on those of the skin by the cold water dash, the application of vinegar to the temples, slapping the palms of the hands or surface generally, frictions with stimulant liniments along the spine,—on those of the stomach, (if the patient can swallow) by a draught of cold water, frequently arouse the heart instantaneously. Æther, aromatic spirits of ammonia, or brandy, should be given internally, if possible, by the mouth; if this be impossible, and the fit be prolonged, enemata, with ammonia, turpentine, or brandy, may be administered. It is scarcely necessary to say, that if protracted syncope depend on overloaded stomach, an emetic should be given (by the rectum, if otherwise impossible): flatulence may be relieved by the rectum-tube, and an asafœtida and cajuput enema.

In cases of protracted fainting, assuming a serious character, hot applications, sinapisms, &c., to the heart and spine, electro-galvanism, and artificial respiration, must be successively had recourse to. If the cause be loss of blood, transfusion presents itself as a final measure.

(*b*.) Where the heart is habitually feeble in action, coldness and clamminess of the extremities, œdema of the ankles even, shortness of breath, frequent inclination to faintness, sensations of languor and ennui; low spirits, anorexia or depraved appetite, foul breath, and constipated bowels, are more or less constant symptoms. The heart's impulse is feeble, its sounds wanting in tone, reduplication of the diastolic sound at the base common, palpitation easily excited.

This state of things, which I have principally seen in young females, and often in connection with disordered menstruation, is curable by attention to the state of the uterus, and by the tonic invigorating plan of treatment. Moderate walking exercise is essential.

Angina Pectoris.

I. Angina pectoris is a paroxysmal disease, of undetermined nature, in which the heart is essentially concerned.

II. Pain, often accompanied with tenderness, in the lower sternal part of the præcordial region, shooting to the back, the left shoulder, and neck, and along the left arm to the fingers, or stopping short at the bend of the elbow, or extending to both arms, or much more rarely to the right arm only, sometimes passing to the left leg, or invading the four extremities at once, and, in rare instances, producing numbness in the testes,—pain of these characters, and perfectly sudden in its onset, is the essential symptom of a fit of angina. Dull, aching in character, lancinating, tearing, or indescribable,—an exquisite torture, constrictive, and suffocative, producing, or certainly coupled with, a dread of impending dissolution, is felt by some sufferers. Respiration is *secondarily* affected; there may be slight dyspnœa or orthopnœa, with lividity of the face,—yet, by an effort of the will (if the patient dares to encounter the pang this commonly produces), the chest may be pretty freely expanded, and the breathing relieved for a brief space: dyspnœa is not a primary phenomenon of angina. The heart palpitates with variable strength and rhythm; the latter may be so disturbed as to produce tendency to syncope: the sounds and palpable impulse vary accordingly. If there be prominently-developed murmurs, there is something more than angina present to produce them. The pulse, as noticed by Heberden, is not necessarily quickened; it may be strong, full, and regular in rhythm (but this, hardly, except in mild seizures), or small, irregular, feeble, and frequent. Convulsive actions may occur; and there may be a full discharge of pale urine.

Lasting a few minutes, or prolonged through an hour, the fit goes as suddenly as it came; or death takes place by syncope.

III. Angina pectoris seems to be constituted by spasm of the heart and neuralgic pain. Whether the pain is mainly, as is ably argued by Dr. Latham, the product of the spasm, or in greatest measure an independent neuralgia, causing the spasm, must be admitted to be an unsettled point. Some reasoners, indeed, doubt the real existence of such a state as spasm of the heart; but the time is not long passed, when the possibility of spasm of the urethra was denied by the schoolmen.—There is not a single structural disease of the heart, of its nutrient arteries, and of the aorta, which has not been found in different victims of angina pectoris. It is, on the other hand, affirmed that death may be produced by angina, the heart and vessels being texturally sound. I doubt this exceedingly: recent narratives invariably describe some organic change, and older accounts are not trustworthy, seeing that dilatation, softening, and various other morbid states have but a few years been understood. It has occurred to me to examine during life some six or eight cases of true angina: in every one there were signs of organic disease. I have opened, or seen opened, the bodies of three persons destroyed in the paroxysm: the heart was texturally affected in all. But, on the other hand, as angina occurs with all varieties of heart-disease, and may be absent with all, (except perhaps very extensive calcification of both coronary arteries,) the conclusion is unavoidable, that there is something (such as just suggested) beyond organic mischief, concerned in generating the paroxysms. That the attack is essentially neurotic (neuralgic or spasmodic primarily) appears from its sudden advent and departure,—from the character and intensity of its suffering,—from the perfect ease enjoyed in the intervals of seizures,—and from the kind of treatment that proves beneficial. The vagus and the sympathetic filaments distributed to the heart are probably the nerves implicated. Why angina should be so peculiarly rare among females, be scarcely observed before the fiftieth year of age, and be comparatively unknown among the humbler classes, as it has statistically been shown to be, does not admit of ready explanation.

IV. Hysterical and anæmic palpitation, accompanied with intercostal neuralgia, nay, even organic palpitation in a person with that form of neuralgia,* may readily be mistaken for true angina; indeed, an attempt has been made by some French writers to show that the disease is nothing more than a "brachio-thoracic neuralgia," the heart-symptoms being purely accidental. In true angina, the points of tenderness in the course of an intercostal nerve are wanting; besides, the severity of the suffering in the cardiac region is infinitely greater than in intercostal neuralgia. Another kind of pseudo-angina is observed in females with neuralgia of the breast and painful palpitation. Palpitation in any form of organic disease may be slightly painful to the sufferer in the same situation, as if he were the subject of angina; is there an imperfect form of that affection actually present?

V. Sedatives and stimulant anti-spasmodics are the medicines essentially to be trusted to during the fit. The dose of opium will be measured by the intensity of the pain; from forty to sixty drops of laudanum or of the liquor opii sedativus may, in a severe case, be given along with from half a drachm to a drachm of *sulphuric* æther or aromatic spirits of ammonia, and repeated according to the urgency of the suffering. Musk, camphor, and belladonna are of very inferior importance. Mustard poultices may at the same time be applied to the heart and to the dorsal spine, or cloths imbibed with the strong liquor of ammonia laid upon the præcordial surface. Laennec's suggestion of the transmission of a magnetic current (with or without acupuncturation) through the chest, has scarcely been fairly tested. An electro-galvanic current, however, affords better chance of successful influence, and in a serious case deserves trial. The mustard pediluvium, especially if the patient be gouty, is useful.

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* *E. g.* Cases of Sus. Roberts and Jane Hawkesford, U. C. H., 1850.

Speciality in the circumstances of the attack may call for special treatment. If the patient be the subject of undoubted sthenic plethora, and especially if the heart be known by previous examinations to be a well-nourished one, the abstraction of blood from a vein, or by cupping between the shoulder-blades, is clearly indicated; but bleeding must not be heedlessly undertaken and without assurance as positive as is attainable, that the heart is at least not a soft and flabby one. If flatulence and acidity exist, soda, cajuput oil, and sesquicarbonate of ammonia may be administered with the opiate medicines; if a large undigested meal lie in the stomach, it should at once (unless the breathing be very seriously embarrassed) be removed by an emetic of sulphate of zinc.

A person who has had one attack of angina pectoris must remember, that instances, in which recurrence does not take place, are altogether exceptional, and further, that the periods of recurrence gradually approximate more and more, and each successive paroxysm, as a rule, exceeds its predecessor in severity.* A first attack is generally brought on by an effort of some kind, such as walking up a hill, or in the teeth of a sharp wind; but eventually the most trivial influence will suffice to produce a paroxysm; emotion of any kind, sudden movements of the trunk or arms, efforts at defecation, the acts of coughing, drinking rapidly, &c. Hence it is clear that the subject of angina must live according to the most stringent rules; every conceivable precaution should be taken to keep the heart in a tranquil state. The patient should give up exciting pursuits of all kinds,—intellectual, corporeal, and emotional,—and learn to govern his temper. Daily exercise should be slowly taken on perfectly level ground, either on foot or in an easy carriage; riding on horseback is scarcely to be permitted with safety. The diet should be moderate in quantity, simple in quality; the bowels never permitted to be confined. A belladonna plaster worn over the heart, and an issue, seton, or perpetual blister to the arm, have appeared useful in some cases: if the patient have confidence in counter-irritation, this should by no means be neglected. Change of scene and travel, coupled with the use of tonics, vegetable or mineral, will, by improving the general health, render the patient less prone to seizures. Nitrate of silver and sulphate of zinc are the best of the class of mineral tonics, unless anæmia be present, when, of course, iron is *the* remedy. The removal of gout, chronic rheumatism, or old standing skin diseases should be very cautiously, if at all, attempted, in the subject of angina: relief of those complaints is unquestionably sometimes followed by increased severity of the cardiac affection.

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* Angina of malarious origin, and recurring periodically, is said to be observed. In such cases the treatment would be that of miasmatic diseases generally.

A paroxysm, of which the too experienced patient learns by his feelings to expect the approach, may sometimes be averted completely by an opiate; and sufferers should always carry (properly protected) on their persons, an antispasmodic and sedative draught.

PASSIVE AND MECHANICAL CONGESTIONS.

I. Passive and mechanical congestions of the heart's tissue and membranes, however interesting to the morbid anatomist, are without clinical importance in the present state of knowledge; there are, in fact, no known means of diagnosticating these states.

II. But congestion of (or, rather, accumulation of blood in) the cavities of the heart is a state at once productive of serious symptoms, and clinically demonstrable. Such accumulation may occur in any, or in all, of the cavities, in cases of endocarditis, as a consequence of fibrinous particles interfering with the free play of the valves,—in cases of polypoid concretions, of whatever origin,—and in cases of rupture of valves. Accumulation in the right cavities, especially, will ensue in prolonged fits of palpitation, with highly-disturbed rhythm,—in cases of tricuspid regurgitation,—during fits of dyspnœa, in highly-marked emphysema of the lung, especially if this has already led to dilatation of the right cavities,—and, probably, to a greater or less extent, in all cases of suddenly obstructed circulation through the lungs.

The symptoms associated with such loading of the right heart are dyspnœa even to orthopnœa, dry cough, venous congestion of the face and upper surfaces generally, unattended (unless there be prior anasarca of the lower extremities) with œdema; oppression, anxiety, and sometimes pseudo-anginal feelings.

The heart's impulse, laboured and struggling, irregular in force and rhythm, is seen and felt more extensively than natural, especially to the right of the sternum, and at the epigastrium. The area of dulness exceeds that of health, especially about the right costal cartilages.

Venesection, cupping over the præcordial region, and sharp, rapid purgation, are the remedies theoretically indicated for the relief of this state; practically, too, they prove useful. But the condition, on which the obstruction of the cavities depends, remains of course in the back-ground, unmodified, or scarcely modified, by them. Ulterior measures must be taken for the removal of that condition, if possible.

PERICARDITIS.

I. Inflammation of the pericardium is clinically known in the acute and chronic forms.

II. *Acute pericarditis.*—The acute disease, in its complete evolution, passes through five anatomical stages,—those of vascularity with dryness, of plastic exudation, effusion, absorption, and adhesion. Each of these stages has its own special physical signs.

1. During the *dry* stage, the extent of visible impulse is greater than natural; the impulse, as felt, is too forcible, of beating rather than heaving character, and successive impulses are of unequal strength. The areas of dulness, both superficial and deep, are unchanged. Grazing friction-sound may occasionally be caught. The physician should, at this period, while it is yet unchanged by the disease, accurately ascertain the point of the apex-beat, in order to substantiate its subsequent displacements.

2. In the *exudation* stage, it is said by some observers, that incipient bulging of the præcordial region may be noticed; muscular paralysis from adjacent inflammation, *plus* the protrusive action of the heart, sufficing for its production. I have never succeeded in discovering this; nor does the theory seem at all sound: admitting that the inflammation of a serous, in close contact with a muscular membrane, paralyses the latter, as in the alleged instance of pleuritis, it does not follow that pericarditis will have any such effect, seeing that the pericardium is not in contact with the side. The inspection-signs are, in fact, the same as in the dry stage. The hand sometimes detects pericardial thrill. If the plastic exudation be very thick, it is conceivable that the area of superficial dulness shall be extended; but this is a point too delicate to be trusted to. The essential sign of this stage is pericardial friction-sound, of which the properties have already been described (p. 229.) The condition of the heart's sounds varies; they may be unchanged, or even louder than in health; or, on the contrary, masked somewhat by the loudness of the friction-sound, or even positively enfeebled, in all probability by the interference of thick layers of lymph with the full play of the ventricles. Valvular murmurs are of

excessive frequency as dependences on co-existent endocarditis,—especially the aortic constrictive and mitral regurgitant varieties. But may valvular murmur come directly of exudation on the pericardial surface? It is conceivable that the aorta and pulmonary artery may be so pressed on by lymph, that murmur shall be engendered with the systole as the blood passes through the slightly constricted part; but I do not know this from experience; in regard of prognosis, the question is obviously one of importance,—a murmur, thus generated, must be of less serious import than one of endocarditic origin. The respiratory murmurs continue unchanged over the heart.

3. The perfection of the signs of the *effusion* stage varies directly as the amount of fluid. By inspection may be discovered arching of the præcordial region, widening and even bulging of its intercostal spaces, with elevation of the left edge of the sternum, sometimes traceably increasing from day to day; œdema of the præcordial integuments, especially if the effusion have existed for any time; undulating impulse; and displacement of the apex-beat upwards as far as the fourth interspace, and slightly outwards. By application of the hand we find that in cases where the apex lies behind a thick stratum of fluid, the impulse lags slightly behind the systolic sound; the impulse feels weak, unequal fluttering, or may be imperceptible; if pericardial thrill had existed, it is now gone; the line of vocal fremitus at the right side of the heart is carried unnaturally to the right (a valuable sign in some cases); the state of respiration-expansion over the heart varies,—if the quantity of fluid be moderate, costal expansion is well marked, diaphragmatic movement being impeded by the fluid,—if very great, that expansion is impaired. The interval between the left nipple and the middle line may be increased; but this is not always the case, even where bulging is well marked. Percussion discloses what is, all things considered, the least fallacious sign of effusion, namely, præcordial dulness of the peculiar pyramidal form and other properties already described (p. 184). The area of this dulness may be

changed sidewards (most readily to the right) by moving the patient successively from one side to the other.

By auscultation, the irregularity of the impulse in regard of force, and, if this be effected, of rhythm, is better perceived than by other means. The friction-sound of the past stage may be either completely gone; or heard in some spots about the great vessels; or pretty *generally* retained in the præcordial region,—but this is very rare even with eight ounces of fluid, and it is scarcely possible with more than ten. On the other hand, *no conceivable amount of fluid will of necessity totally annul friction-sound.* I base this statement on a case in which I and others distinctly heard friction-sound "at mid-sternum on the level of the third rib," and yet (death occurring only twenty-nine hours later) sixty ounces of fluid were found in the pericardial sac, which reached about a thumb's breadth *above* the clavicle.* The possibility of systolic basic murmur being produced by pressure of fluid on the great vessels has already been considered (p. 216). The heart's sounds feeble, distant, and as it were muffled, at the lower part of the cardiac region, become louder as the stethoscope is carried upwards, and at the top of the sternum the second sound is full and clear, and the first very decidedly more marked than directly over the ventricles.

The anterior edges of the lungs are pushed aside by the accumulating fluid; the central tendon of the diaphragm undergoes depression, and may be rendered convex inferiorly; the liver *may* be pushed downwards and to the right, but an enormous amount of fluid will not necessarily displace it.

The respiratory murmurs in the centre of the cardiac region are feeble and distant: in some very rare instances the voice resounds with an ægophonic twang at the edge of the effusion: this is especially likely to occur, if the adjacent border of the lung be indurated.

4. In the stage of *absorption* undulatory impulse disappears; the point of the apex-beat falls (not invariably, however); the

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* Case of Bartlett, U. C. H., Males, vol. iv., p. 292.

bulging of the cardiac region gives way. By the hand we ascertain that the impulse has recovered its breadth and fulness; friction-fremitus, too, may return. The dulness of effusion gradually diminishes from above downwards, and draws in laterally also, but not till it has undergone a very distinct fall superiorly. Redux friction-sound is caught by the ear, commencing about the roots of the vessels, and varying in extent with the rapidity of absorption. The churning or continuous rumbling variety (p. 230) is the rarest quality of sound discovered. The heart's sounds recover their fulness (if endocarditis have not prominently existed), and also their natural nearness to the surface; the respiration returns slowly, and may never, especially if agglutination of the pleural surfaces occur in front of the pericardium, recover its natural intensity.

5. The occurrence of the last stage, that of *adhesion* of the pericardial surfaces, is announced by disappearance of friction sound, and *à fortiori* of friction-fremitus, if this have existed; the former may continue audible in some points, where adhesion is as yet unestablished. The percussion dulness continues to decrease, or at all events does not increase, in area. The action of the heart may be tremulous, unsteady, or jogging.

The moment adhesion is accomplished, the evolution of the acute disease has reached its final term.

III. The chief local *symptom* of pericarditis is pain, occupying the cardiac region only, mainly seated in the epigastrium, or extending to the left shoulder and elbow,—slight in amount or of agonising severity, lancinating, tearing, burning, or consisting of a mere sense of soreness; and increased by movement, and deep inspiration. Pain, however, may be absent, and in the majority of cases is either absent or of slight severity. The varying amount of suffering in different cases is with difficulty accounted for; coexistent phrenic pleurisy seems to explain the acute agony of some patients, but pleurisy *may* be present and the pain moderate, or *vice versâ*. The intercostal spaces are generally tender; pressure in the epigastrium causes great distress. Palpitation is not usually a prominent symptom, and may be totally deficient, especially as a condition of which the patient is painfully conscious.

The decumbency is least commonly on the left side, most commonly on the back; the head is generally kept rather high. Orthopnœa is a most inconstant symptom; if present, it is not any proof (as has been taught) that liquid effusion has taken place; it may be absent during the effusion-period, and first appear after absorption; again, it may occur paroxysmally and irregularly. Far from orthopnœa being a necessary result of effusion, the patient, where this is most copious, may lie by choice flat on the back, with scarcely any pillow: this was the habitual posture, for days, of the patient Bartlett, just referred to. In such cases, the least elevation of the head produces a tendency to syncope,—and the patient instinctively dreads movement of any kind. The face is generally anxious; the features drawn; in fatal cases, *risus sardonicus* sometimes occurs towards the close. The sleep is fitful and disturbed; and jactitation of the arms (the trunk being kept quiet) is not unfrequent in serious cases. Rigors may announce the invasion of the disease, but they are not commonly severe nor repeated; the skin is subsequently hot; if perspiration occur, it is not specially acid, unless rheumatism be present,—and it *may* be alkaline, even with this combination. Sudamina sometimes form, and their contents may be alkaline, while the surrounding perspiration is acid,—or the reaction of both may be the same. Œdema sometimes occurs, particularly if the case be protracted, about the ankles; and may also appear in the integuments of the cardiac region. The integuments of the head, face, and neck, may become markedly livid. The joints are not affected as a consequence of pericarditis,—they are frequently rheumatic, of course, as the majority of cases of pericarditis are of rheumatic origin. The limbs generally are the seat of febrile pains. There is nothing special in the state of the tongue. Spasmodic dysphagia sometimes occurs. Tenderness of the epigastrium, with nausea and vomiting of food, or of bile even (the stomach being perfectly sound,*) sometimes constitute very prominent symptoms. The liver grows engorged; I have never seen jaundice. Dry, irritable, abrupt, jerking, spasmodic cough, with variable dyspnœa, and coolness of the expired air towards the close, are the chief pulmonary symptoms: that exaggerated respiration results from pericarditis *per se*, I more than doubt. The pulse is frequent out of proportion with the respiration, unless there be some pulmonary complication: at first full and hard, afterwards weak and feeble, the pulse grows irregular, both in force and rhythm, in about one-third of cases,—or such irregularity may exist from the first, and before fluid is present to account physically for the circumstance. The frequency of the pulse is subject to more sudden variations, from the influence of effort, than in any other disease perhaps: thus I have known a very gentle movement of the trunk raise the pulse from 80 or 90 to 130 or 140. The blood is hyperinotic. The urine is febrile, or possesses the characters of the diathetic disease, with which the pericarditis is associated. Cephalalgia, though present, is not often spontaneously complained of, and all serious head symptoms may be totally absent from first to last. But, on the other hand, delirium, apoplectiform stupor, and a quasi-maniacal state sometimes occur. So, too, serious symptoms of spinal character (produced in some cases by reflex irritation from the pericardium, in others by uræmia) occasionally appear: as cramps, epileptiform, hysteriform, and tetanic seizures, and chorea† in all degrees of severity.

IV. The terminations of acute pericarditis are by recovery, by death, or by the chronic disease. Complete recovery, anatomically speaking even, occurs sometimes by resolution, and also by absorption, without consequent adhesion. Death may happen suddenly through the mechanical obstruction of the heart's action by the accumulated fluid,—but it is wonderful what an amount may be borne without this result. Sudden death may be caused by movements of the trunk; where previously the case had been proceeding most favourably,—a fact never to be lost sight of by the practitioner. The characters of the chronic disease will by and bye be considered.

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* Case of G. Perry, U. C. H., Females, vol. i., p. 82, 1846.

† Chorea in childhood I hold to be a most ominous complication: of four well-marked cases of the kind, which I have seen within the last two years, three terminated in death. In all four the pericarditis was rheumatic.

V. Pericarditis may be of traumatic origin; be produced by the irritation of local morbid formations (tubercle, cancer); result from the extension of neighbouring inflammatory action (pneumonia or pleurisy); appear as a sequence of phlebitis and pyohœmia; occur as part of certain diathetic affections (rheumatism, gout, and Bright's disease); or, in very very rare instances, be purely idiopathic. Alleged idiopathic pericarditis becomes rarer every year, in proportion as the evolution of diathetic diseases grows more fully understood; I have never seen a case of pericarditis of the kind.

VI. The diagnosis of pericarditis can only be made with security by the physical signs; for the disease may be absolutely *latent* from first to last (I have known patients with several ounces of fluid and exudation-matter in the pericardium, grow irritated, when inquiries were made about symptoms connected with the heart); or there may be a total want of harmony between the violence of the symptoms of which the patient is actually conscious and the amount of disease; or the symptoms of other affections may be simulated.

The essential signs of pericarditis are friction-sound, special præcordial dulness, and twisting upwards of the heart's apex. Friction-sound, when thoroughly developed with the characters assigned it elsewhere (p. 229), is next to pathognomonic; the possible fallacy from pleural friction of cardiac rhythm (p. 235) must not, however, be forgotten. Mediastinal pseudo-rhonchus, and the squashy rhonchoid sound, sometimes produced over the heart, when the integuments are œdematous, are other possible sources of difficulty. The rules already laid down

(p. 236) will distinguish friction from endocardial murmur. On the other hand, pericardial friction may really be heard, and mistaken for other things. The clicking variety differs, however, from valvular clicks in its non-synchronism with either heart-sound, and in its non-transmission along the aorta. Again, exudation may be present, and yet no friction sound evolved,—either because the posterior aspect of the heart only is affected,—or because one lamina of the membrane only is coated with lymph,—or because recent agglutination has occurred,—or because old agglutination prevents attrition,—or because effusion has been poured out.

As concerns percussion-dulness, if the peculiar pyramidal dulness be developed under the eye of the physician, and have succeeded to friction-sound, there is no possible source of fallacy. But if it have not been preceded by friction, the dulness may be from hydropericardium simply; and if the pyramidal form have not been produced under observation, there are certain sources of fallacy of a serious character already mentioned (p. 186). Thus a weak fatty heart, with quasi-undulatory impulse and feeble sounds, intermittent pulse, and febrile action, may exist, where the form of the heart's dulness is rendered triangular by the chance presence of old exudation-matter about the great vessels, a tumor, a small solid quiescent aneurismal sac,—or even of an excess of natural mediastinal fat. How is the distinction of the cases to be established? Probably, in the whole range of thoracic diagnosis, there does not exist a more difficult problem. Thus, an adult labouring obviously under acute disease, unable to give any trustworthy account of himself, had all the physical heart-signs mentioned, and in addition orthopnœa and jactitation, while, on his admission to the hospital, no ordinary signs of pneumonia or other thoracic inflammation existed to account for the acute aspect of the disease. I was strongly disposed to regard this as a case of latent pericarditis, with effusion (accompanying valvular disease, of which the signs were obvious); but the impossibility of discovering a shadow of friction-sound, though the posture was varied, the non-elevation of the heart's apex, and the fact that above the third rib the dulness was not so absolute as below it, led me to reject the idea. On *post-mortem* examination (the signs of pneumonia had meanwhile made their appearance,—on admission, indeed, the pulse-respiration ratio was 2·7 : 1, and in twenty-four hours had become 2 : 1), the pericardial sac was found free from fluid; but above the base of the heart lay a lump of fat,* the simple source of all the difficulty.

Mediastinal tumor of some size, if it encroach on the cardiac region, may simulate pericardial effusion: the history of the case; the presence of concentric pressure-signs, which are never caused by fluid in the pericardium; and the outline of the dulness, which can scarcely by an unlucky chance imitate precisely that of pericardial dulness, (a tumor may grow in spite of gravity, fluid obeys this,) will distinguish the former from the latter.

Here is another puzzling combination: a female may have fever, dry red tongue, extreme epigastric, and no præcordial, tenderness, bilious vomiting and diarrhœa, and perfectly regular pulse,—present not a single symptom connected with the heart, and be free from Bright's disease or acute affection of the joints. How is this state of things, really dependent on pericarditis with effusion, to be distinguished from acute gastritis? The physical signs say nothing: friction-sound is gone, when the patient is first seen, and a huge stratum of subcutaneous fat and a massy mamma may deprive us of the evidence of percussion.†

In the diagnosis of a difficult case, the functional and general symptoms should not be forgotten,—but clinical experience compels us to admit that they are sometimes utterly fallacious aids. Hope wrote the singular proposition, that "the variability of the symptoms is calculated rather to enlighten than perplex the practitioner,"—his belief being that the symptoms of lymph-deposit were slight, of effusion serious. The value of this dogma appears from a single case, the fourth in Andral's Collection, where *death* occurred from plastic pericarditis in twenty-seven hours, without a drop of *fluid* having formed in the sac.

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* Case of Beckett, U. C. H., Males, vol. v., p. 229.

† All these facts were illustrated by the case of E. Perry, U. C. H., Females, vol. i., p. 82.

VII. The *treatment* of pericarditis will in some wise be modified by the diathetic state it accompanies. If it appears in the course of rheumatism (either after, along with or before the joint affections), it is possible the intensity of the inflammation may be lessened by artificially irritating the joints. To grant this, obviously does not require one to have any faith in the exploded doctrine of metastasis; but the *practice* has disappeared (perhaps unmeritedly) since the *theory* has been banished from the schools.

Bloodletting takes the first rank among the remedies in pericarditis; but in judging of its utility in any particular case, the causes of the inflammation must be borne in mind: rheumatic pericarditis in the great majority of cases terminates favourably, no matter what be the precise mode of scientific treatment adopted; pericarditis of renal origin, almost as invariably proves fatal. Bleeding from the arm is attended with a certain amount of risk of syncope; it does not prevent other inflammations coming on, be it ever so free; very copious depletion in certain constitutions excites the heart greatly, and in some respects makes matters worse; and the most severe rheumatic pericarditis (variously complicated) *may* go calmly on to recovery, though cupping over the præcordial organ has been substituted for venesection.* Whether bleeding at the outset may ever arrest the disease at once, I do not believe to be scientifically ascertained: it may lessen pain and distress without a jot abating the activity of the exudation-process. But, *per contra*, that bloodletting shortens the duration of pericarditis, and does so the more effectually the earlier it is performed, has been clearly shown by Dr. Taylor in his logical papers on the treatment of the disease.* The quantity of blood to be drawn must be regulated by the severity of the symptoms: from an adult of medium strength (it is to be remembered that loss of blood is worse borne in renal than in rheumatic pericarditis) some twelve to sixteen ounces may be taken from a vein in the arm, the head being kept *low*, especially if there be much fluid in the sac. This depletion may be followed, if well supported, by the abstraction of some six or eight ounces more by cupping or leeching over the heart. In cases of slight severity, local bleeding may be most confidently trusted to alone.

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* *E. g.* Case of Craddock, Clin. Lect., loc. cit.

The accurate evidence before the profession concerning the influence of mercury, though it leads us to question the extreme power of the mineral, nevertheless shows that its administration is not to be neglected. It stands obviously second to blood-letting,—and appears to carry out, as it were, the good effects produced by this. Salivation is with difficulty induced, as is well known; although no positive proof exists that the utility of the mineral is measurable in this disease by the rapidity of ptyalism, still, in obedience to general conviction, it is well to ensure this result as speedily as possible; and this may best be done by the plan recommended in a previous page (p. 298). Ptyalism being effected, the disease is not necessarily arrested; Dr. Taylor refers to three cases in which increase of the disease distinctly followed.†

On the principle of regarding the disease through its diathesis, colchicum and alkalies are advisable, where the pericarditis is rheumatic; I do not think colchicum should ever be omitted in a case of the kind, and it may be given in the form of draught along with the mercurial pill. Opium becomes a necessary remedy in full doses, if the agitation and disquietude be at all marked; morphia may be used endermically, if it has been found advisable to apply a blister to the præcordial region. The application of a blister is, however, objectionable from its interfering with the proper examination of the cardiac region, and hence preventing a precise knowledge of the state of the disease; blisters should rather be applied behind, than actually on, the præcordial region. Digitalis, aconite, and hydrocyanic acid are dangerous agents, from the chance of their increasing tendency to syncope. Purgatives, diuretics, and diaphoretics are advisable as aids in the treatment.

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* "Medical Times," 1851.

† Dr. Taylor's evidence, indeed, so far as it goes, rather bears against mercury. He refers to the frequent occurrence of acute inflammations during salivation for the cure of others. I have known pericarditis supervene in a woman while under treatment for ptyalism, so severe, that for some hours after her admission into hospital for the cure of that ptyalism (Spratt, U. C. H., Females, vol. iv.. p. 471) life was in danger from semi-asphyxia; but such cases must be esteemed singular exceptions.

Sinapisms frequently repeated are of great service in relieving pain and distress, and are not open to the objection just mentioned in the case of blisters. Ioduretted frictions, coupled with mercury in very small proportion, seem to promote absorption of exudation-matter.

The regimen must be strictly antiphlogistic. Of the secondary morbid states that occasionally ensue on pericarditis, namely, endocarditis, carditis, local pleurisy, pneumonia, bronchitis and chorea, the latter is the only one requiring specific measures. The occurrence of chorea (which is, however, rare at the outset) is an indication for the total suspension of depletory measures and of mercury; purgatives, antispasmodic and sedative remedies, must at once be had recourse to.

VIII. *Chronic Pericarditis.*—Under the term chronic pericarditis may be included two states clinically very different,—namely, that in which effusion remains in the sac without apparent inclination to increase or to disappear,—and that in which adhesions or agglutinations of the pericardium having formed, a tendency to active congestion in the pericardium itself, and in the substance of those adhesions, is more or less constantly present. The former condition is rare, the latter common.

(*a.*) To begin with the case of adhesions. We have seen what the signs of adhesions are at the time of formation; they are easily established. But in a case, seen for the first time, after adhesions have been some time formed, their positive diagnosis is among the most difficult clinical problems existing. Their signs vary with the size of the heart, the state of the valves, the closeness of the pericardial adhesions, and *the presence or absence of pleuritic adhesions in front of the heart.* All I can say on the matter is as follows:—

The apex-beat sometimes remains fixed in the high position to which it was raised by the effusion-period; but more frequently hypertrophy, sequential to the pericarditis, carries the mass of the heart downwards. Respiration has less effect than in health in lowering the organ. Permanent depression of the præcordial region, superiorly or inferiorly, may exist. The epigastrium may dimple inwards with the ventricular systole, and the lower part of the sternum and adjacent cartilages be drawn slightly inwards at the same time; but this I have never seen, *unless the pleural surfaces in front of the heart were closely adherent.* The respiration-movement is very limited in the præcordial region. Undulation I do not happen to have observed,—but it is said by some observers to exist in these cases. There is frequently a certain amount of dulness above the third cartilage, from the presence of false membrane about the great vessels. A jogging throbbing motion of the heart has been ascribed to the influence of pericardial adhesions: it rather depends, when it exists, which is on the whole rare, on co-existent hypertrophy and dilatation of the organ. Friction-sound may long continue after *partial* adhesions have formed; complete agglutination will, of course, put an end to it: the variety I have described as *clicking* (p. 230), may sometimes be caught. It has been said that feebleness, even to extinction, of the second sound, is a sign of pericardial adhesion: I doubt whether the two things, when associated, have ever any direct connection; and I know that complete agglutination may co-exist with a perfect second sound.

If new effusion occur in a formerly inflamed pericardium, the continuance of respiration in the præcordial region is a sign of adhesion, pericardial and adjacent pleuritic combined.* A past pericarditis does not prevent the characteristic signs of effusion occurring a second or a third time, provided there be not complete agglutination; friction-sound, pyramidal dulness, and twisting of the heart's apex upwards may all be present.

If an adherent pericardium be the seat of irritative action, local depletion, blistering and liniments stimulant and absorptive, to the cardiac region, are the most important remedies. Iodine should be given internally. But the chief evil to contend with is generally hypertrophy and dilatation of the ventricles, mainly the left: where agglutination exists, this mode of enlargement of the heart almost invariably commences within a short period. But I confess that my observation does not lead me to take the very gloomy view expressed by some writers of the ultimate issue of such cases: I have not, as they appear to have done, seen pure chronic adhesive pericarditis prove rapidly fatal. The question is, of course, a very different one if there be valvular disease superadded. An atrophous state of the heart appears sometimes to follow from the tight embrace of pseudo-membrane on its surface.

(*b.*) The signs of fluid stagnating in the pericardial sac are those of the effusion-period. If these signs remain unchanged by ordinary means of treatment, (hydragogues have little effect,) paracentesis of the pericardium becomes justifiable, as an *ultima spes*, provided urgent suffocative symptoms exist. The patient is certainly not placed in a worse position by the operation, than he was before it; the immediate relief is extreme, and a certain very small chance exists of at least temporary recovery. Were the operation determined on, a trocar should be cautiously introduced, perpendicularly to the surface, at the lower angle of the left fourth interspace close to the sternum. The fluid, which escapes by jets, corresponding to the ventricular systoles, should be evacuated as completely as possible before the wound is closed, —a syringe may even be employed to ensure this. But the orifice of the canula, before its removal, should occasionally be closed, lest too rapid removal of the fluid might produce evils of its own on the heart, accustomed as this has been, for a greater or less time, to considerable pressure.

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* Case of Craddock, loc. cit.

ENDOCARDITIS.

Acute Endocarditis.—I. In acute inflammation of the endocardium, the heart's movement is seen and felt to influence the surface more extensively, more forcibly, and more abruptly than natural. The surface is not bulged, and the point of the apex-beat not (as in pericardial effusion) raised upwards; it may be carried a little downwards and outwards even; there is no undulatory movement, and no tactile thrill. Hope says he has observed the thrill of mitral regurgitation, but he gives no proof that the regurgitation was purely recent: I have not succeeded in finding thrill when any certainty existed of the absence of old-standing mitral disease. The areæ of the heart's dulnesses, both superficial and deep-seated, undergo increase; the former because greater energy of action brings the heart more uniformly forwards, the latter because the walls of the organ are turgid, and its cavities more or less clogged with blood. The area of dulness is never seriously increased, unless there be considerable distension of the heart by accumulated blood.

Auscultation discovers a murmur or murmurs, blowing in quality, soft and low-pitched. The murmurs of *purely acute* endocarditis may, as far as I have observed, be thus arranged in order of frequency :—aortic obstructive; mitral regurgitant; aortic regurgitant; aortic obstructive and mitral regurgitant together. I have never observed *acute* obstructive mitral murmur, nor *acute* regurgitant tricuspid murmur; the latter, especially, I believe to be at the least very rare,—a circumstance in accordance with the fact that most chronic tricuspid regurgitant murmurs are produced by simple incapacity of the valve to fill the widened orifice, without actual disease of its own tissue.

The site and rhythm of acute endocardial murmurs, it is supposed, may change during the course of an attack;—lymph, it is presumed, may be absorbed or washed away and deposited elsewhere, and a different species of murmur consequently developed. I have not observed this; but I have known systolic aortic followed by diastolic aortic murmur, apparently from an increase of lymph.

The murmurs of *acute* endocarditis are produced by roughness on the surfaces, by intertwined lymph (or mere fibrine) interfering with the play of the chordæ tendineæ, or, probably, by non-closure of orifices through irregular action of the papillary muscles.

Such heart-sounds as are not replaced by murmurs, present no constant character. Reduplication of the second at the base is common. Probably, at the outset, they are both intensified; and murmurish prolongation of the first, before it actually becomes a murmur, is sometimes noticeable.

In cases where the circulation through the heart's cavities is obstructed seriously, either from accumulation of lymph and fibrinous coagula, or from rupture of a valve or chorda tendinea, the impulse becomes irregular in force and rhythm,—at first violent, subsequently feeble; the heart's dulness extends notably, especially to the right of the sternum; the sounds or pre-existing murmurs are enfeebled, or new murmurs may be generated. The suddenness of occurrence of these signs points to their source.

II. The local symptoms of endocarditis are not very marked. Pain is rare, discomfort and uneasiness at the heart common; more or less palpitation exists; tenderness of the præcordial interspaces is at the least unusual, unless there be co-existent pericarditis.

The general symptoms vary with (*a.*) the free, or (*b.*) obstructed state of the circulation through the heart. (*a.*) The decumbency is generally dorsal, the attitude quiet; but jactitation of the arms occurs in some cases. The skin of febrile heat, the integuments unchanged in colour; the joints rheumatic, or unaffected; no special sensation of dyspnœa is complained of; the respiration holds its natural ratio to the pulse, so long as the orifices are not seriously obstructed, and no secondary pneumonia has occurred; sometimes a little dry cough exists without bronchial or other rhonchi; the pulse is not remarkably accelerated, ranging between 80 and 120,—Dr. Taylor's statement that it loses in frequency at the outset of endocarditis, I have not had an opportunity of confirming. The blood is hyperinotic in the sthenic disease; if the inflammation be the effect of phlebitis, pyohœmia, &c., the clot is soft, but little or at all buffed: endocarditis may also, in all probability, secondarily, cause the latter condition of the blood by the circulation of its own inflammation-products. The urine is simply febrile. Cephalalgia exists commonly more or less,—slight wandering may occur at night, but otherwise the head remains free. I once saw acute mania occur during the convalescence of endocarditis (rheumatic and without pericarditis).* Choreal symptoms are not induced, if the disease remain simple.

(*b.*) In the obstructive class of cases, the action of the heart, suddenly at the moment of obstruction, becomes excessively frequent, uneven, and irregular; the pulse small, weak, irregular in force and rhythm, mounts to 130, 140, 160, or even more. Semi-syncope, pallor, coldness of surface, anxiety, and jactitation, inclination to orthopnœa (which the patient resists from its increasing faintness,) with, eventually, the symptoms of complete pulmonary obstruction, lividity of surface, turgescence of the face, prominence of the eyeballs, puffiness of the ankles, supervene,—the brain suffers also congestively, as exhibited by fitful snatches of sleep, convulsions, delirium, and somnolence, lapsing into fatal coma. I have seen these symptoms in a minor degree, and passingly, in certain cases of endocarditis, which terminated favourably,—in all probability, in those instances, small concretions had formed, and subsequently undergone disintegration and solution. The symptoms of rupture of a chorda tendinea during the acute disease, are extremely similar;—the effects on the cardiac circulation must, indeed, be closely analogous.

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* Case of Cooper, U. C. H., Males, vol. i., p. 129.

The blood, in certain cases of endocarditis, receiving the products of the inflammation, becomes spoiled. Rigors, heat of skin, and profuse perspiration recurring irregularly, dull, earthy, yellow discoloration of the skin (not of the conjunctivæ,) diarrhœa, more or less bilious, pinched, anxious countenance, intense prostration, and muttering delirium, announce this occurrence; and are followed by the evidences of secondary nodular pneumonia or hepatitis.

III. The terminations of acute endocarditis are—(1.) Very serious valvular disease, followed by implication of the heart's substance, and all their combined consequences: the ensuing affections of the heart are, in their order of frequency, eccentric hypertrophy, simple hypertrophy, simple dilatation, and, in infinitely rare cases, eccentric dilatation.* (2.) Slight valvular disease, with habitual palpitation. (3.) Slight valvular disease, with palpitation under excitement. (4.) Simple murmurs, without any positive cardiac functional disturbance; no morbid palpitation occurring even under severe exercise: this is the most favourable result observed; an indubitable endocarditic murmur never, as far as I have known, totally disappears.† (5.) On the other hand, death is rare from acute endocarditis alone; still, the disease does occasionally kill, both by secondary impregnation of the blood, and by seriously obstructed circulation through the heart.

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* The relationship of the part of the heart affected to the disordered orifice, will be considered with valvular disease generally; for the endocarditic origin of the valve-disease gives no *special* character in this way.

† Temporary obscurity of a recent mitral murmur sometimes comes of the weakness of the heart attending convalescence, and leads to the idea that the murmur is gone, or will go, completely; as the general vigour improves, the murmur recovers its distinctness.

IV. The diagnosis of acute endocarditis is essentially based on the existence of febrile action, cardiac uneasiness, excited action of the heart, and endocardial murmur,—this murmur occurring in a person presumedly free from prior cardiac disease, and presenting no other obvious acute affection to explain the febrile action.

But a murmur, existing when a patient is first examined, may be new, or it may be old. Now granting that it is new, it may not be the product of endocarditis. For the excitement of the heart may depend on some other, latent, inflammation or as yet unevolved disease; and this excitement, coupled with a modified state of the blood, may suffice to generate a murmur. Thus, occasionally, at the outset of pneumonia, of the exanthemata, and of any inflammatory state, in persons whose blood chances to be in the least spanæmic, murmur occurs. But such murmur, purely of blood origin, is necessarily systolic at the base; if there be new murmur at the apex, or of diastolic rhythm at the base, a complication of acute endocarditis must be admitted; if the murmur be basic and systolic only, the diagnosis must be deferred.

So far the murmur has, for argument sake, been admitted to be new; unfortunately the great difficulty is often to determine that it *is* new. Now two cases present themselves here: (*a*) the murmur exists, when the patient is first seen; or (*b*) it is developed, after observation of him has commenced.

(*a*) A murmur being already present, the circumstances, *within itself*, favourable to recency of origin, are softness of blowing quality, lowness of pitch, systolic rhythm and aortic constrictive or mitral regurgitant mechanism. The circumstances hostile to recent origin are roughness of quality, high pitch, diastolic rhythm (indeed this is absolutely conclusive, unless there be systolic murmur at the same orifice) and seat at the tricuspid orifice; direct mitral murmur, also, I believe, is never recent.

The presence or absence of affections with which endocarditis is commonly associated, furnishes a guide not to be despised; but without caution the observer may readily be led into error, as in acute rheumatism, by too implicit trust in this very guide. The condition of the pulse cannot be confided in for diagnosis.

(*b*) An endocardial murmur, *developed under observation, at the early period of an acute attack*, is almost a sure index of endocarditis; but even here there are sources of fallacy. In the first place appear those just enumerated, on the hypothesis of the murmur being by admission new. In the second place, general collapse and failure of the heart's power may prevent a murmur from being heard, of which the physical chronic conditions exist in perfection. Reaction takes place, and a murmur becomes audible; that murmur may be chronic murmur solely, or it may be an acute *plus* a chronic murmur; but it is *not* that, which it would alone seem to be, namely, an acute murmur *solely.** Again, to have value as a positive sign of endocarditis, the murmur must be developed at an *early* period of acute disease; if towards the close, it is generally a consequence of spanæmia.

If during the course of an acute febrile disease, endocardial murmur changes in site and rhythm, this is a very strong, probably an absolute, sign of its dependence on recent inflammation. But such change is at the least very rare. Again, if a murmur of a certain site and rhythm disappear and return within a short period; this would appear proof positive of recency of origin of the cause of the murmur; but it is not so, at least of all varieties of murmur. I have known a purely organic direct mitral murmur come and go in this way (p. 226).

V. The treatment of endocarditis is essentially the same as of pericarditis, and active measures are obviously called for, as the ultimate dangers of valvular disease are extreme. Blood-letting and mercurials are the main agents.

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* Case of Kernis, U. C. H., Females, vol. ii., p. 237. The statements in the text are made on clinical and *post-mortem* evidence.

When the affection seems lapsing into the chronic state, iodide of potassium and liquor potassæ, with bitter tonics, become the best remedies; and iodine-inunctions over the cardiac region seem occasionally useful.

If there be reason to suspect the formation of polypoid concretions, the rapid pouring in of liquor potassæ seems, theoretically at least, worth trial; if there be sinking tendency, sesquicarbonate of ammonia may be given at the same time. Sinapisms should be applied to the extremities. I have seen temporary relief obtained by cupping over the heart; but the patient's strength is rarely, when such obstruction occurs, in a condition to bear the loss of even a few ounces of blood.

Chronic Endocarditis.—Chronic endocarditis is solely known clinically by its effects on the valves and orifices of the heart, and there is nothing in the physical characters of valve-disease produced by inflammation, distinguishing it from that produced by other causes. I therefore refer the reader to the section on valvular disease in general.

CARDITIS.

I. The anatomical characters of *acute* carditis, as an attendant on endo-pericarditis, are well known; they are of frequent occurrence, on a limited scale, in the strata of fibres nearest the inflamed membranes. But clinically, the effects of such carditis are not understood; whatever they are, they are lost in the more striking phenomena of the membranous inflammations; possibly great weakness and fluttering character of the pulse may sometimes be due to inflammatory softening of the left ventricle, in cases of pericarditis without much fluid effusion.

II. As an idiopathic separate state, general carditis is clinically yet more unknown. As far as I know, there are not more than half a dozen cases of the kind on record, and the narratives of these furnish no guide to the detection of the disease during life. The disease has yet to be *observed*, before its description can be written.

III. Partial carditis sometimes occurs, producing abscess, ulceration, and rarely actual gangrene; but of these states no positive clinical signs are known. If perforation or rupture occur, as they sometimes do, the symptoms will vary with its direction; if the septa be perforated, sudden cyanosis may follow, or, it is alleged by Bouillaud, no particularly serious symptoms ensue. The effects of rupture of the heart into the pericardium will be elsewhere considered.

IV. Shortening and thickening of the papillary muscles, and infiltration of their substance with induration-matter, due to a *chronic* inflammatory process, derives its interest from its interference with the closure of the tricuspid or mitral valves; but there is no character in either a regurgitant, mitral, or tricuspid murmur, distinctive of this special mechanism.

CARDIAC HÆMORRHAGES.

I. *Of the Muscular Substance.*—Hæmorrhage in this site may form specks, or apoplectiform nodules of blood; or appear in the infiltrated state in connection with (rather, perhaps, as effect than cause) local softening. Under the latter circumstances, rupture or perforation of the heart may occur, and produce fatal extravasation of blood into the pericardium. Such mode of death might be suspected in an individual suddenly cut off with signs of accumulation in the pericardium, and previously known to have a weak heart and to be free from aneurism of the aorta: but there are no positive symptoms of hæmorrhage limited to the heart's actual substance.

II. *Of the Pericardium.*—(*a.*) The effusion of inflammation is sometimes so much stained with blood, as to entitle the disease to the name of hæmorrhagic pericarditis. It seems probable that such escape of the blood-disks does not take place from the vessels, unless the constitution of the blood itself be affected. I have seen this variety of inflammation well marked in pyohæmia. But no signs or symptoms are known, whereby the hæmorrhagic addition to the effusion might be recognised during life. Neither is there any evidence to show that, in the event of recovery, the material of adhesion will be of different character from that observed in the more ordinary class of cases. The quantity of blood is sometimes very considerable,—quite enough to give a deep red colour to all the fluid in the sac: were paracentesis performed in such a case, the operator would very probably be led, for a moment, to believe that he had punctured some important vessel.

(*b.*) Hæmopericardium may be caused by wounds or by ruptures of the heart itself, by rupture of an aneurism of the aorta or of the heart, by rupture of a coronary artery or vein, or by the giving way of cancerous substance. In all these cases the result is almost instantaneous death, from mechanical obstruction of the heart's action. Some of these cases will again be referred to; the rest are devoid of clinical interest.

(*c.*) The pericardium is the seat of extravasation of blood in cases of scurvy,—especially in some localities; in certain parts of Russia, scorbutic hæmopericardium seems as thoroughly endemic as hæmaturia in the Mauritius.

It occurs with or without previous scorbutic symptoms, and the attack may be sudden, or so gradual that attention is scarcely drawn to the heart. Præcordial oppression, without pain, or tenderness, and great dyspnœa, seem to constitute the main symptoms; the physical signs will, of course, be those of a pericardium distended with fluid.

The more frequent termination is by death; but recovery sometimes takes place by absorption of the blood,—the anatomical conditions remaining, being very assimilable to those of chronic pericarditis. Indeed it seems highly probable that the disease is from the first sub-inflammatory.

The treatment, locally, is by cupping,—generally, that of the blood-disease present. In a large number of cases in which paracentesis was performed by Russian physicians, the ultimate result was unfavourable; but in all the immediate relief was extreme,—the patient seemed temporarily endowed with new life; and two cases are given of complete recovery.

DISEASES OF SECRETION.

Hydropericardium, or dropsy of the pericardium, may be of active, passive, or mechanical origin.

(*a.*) Active hydropericardium is very rare: I have, however, in some instances of Bright's disease, known the pericardium fill with fluid,—the symptoms indicating an irritative state, while the signs of pericarditis were wanting. I once saw a case which suggested to my mind the question, whether in true hydropericarditis, the plastic material might not be completely absorbed, and the serosity left behind,—constituting a sort of sequential active hydropericardium. But I have no positive answer to supply; and, possibly, the case referred to was one of active dropsy alone from the first. When hydropericardium is active, it may be the sole dropsy in the body.

(*b.*) Passive hydropericardium occurs as a phenomenon of general dropsy,—very rarely unless double hydrothorax be already present. The quantity of fluid (colourless, straw-coloured, or slightly blood-stained, but without lymph) is generally moderate,—from eight to twelve ounces; I have never seen more than the latter quantity.*

(*c.*) Mechanical hydropericardium has, in some very rare cases, been traced to pressure of carcinoma on the great veins, and to morbid states of the coronary veins.

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* A case recorded by Corvisart (Maladies du Cœur, 2ème édit., p. 52), where "about four pints, or eight pounds, of clear greenish serosity," were found in the pericardium, seems to have been one of chronic pericarditis. But on any hypothesis, the quantity is almost marvellous, and throws completely into the shade that measured in my patient, Bartlett (p. 441).

Hydropericardium, however originating, has few subjective symptoms: acute pain and tenderness are altogether, and palpitation commonly, wanting; sensations of weight and oppression are alone complained of. The physical signs are in the main those of hydropericarditis: but there is no friction-sound, no præcordial bulging, and the apex of the triangular dulness (simply because there is less fluid) does not mount so high as in many cases of pericarditis,—the impulse may be very perfectly undulatory.

The general symptoms are those of the disease on which the local dropsy depends. The pulse is not necessarily irregular; the quantity of fluid is not commonly sufficient to affect the heart's action very seriously. Orthopnœa may exist; but if so, as far as I have seen, there is double hydrothorax to share in its production.

Diuretics and hydrogogues seem to have less effect on this dropsy than on others: such medicines will, however, of necessity, be tried, were it only for the usually concomitant dropsies. Cautious cupping, or dry-cupping over the heart, would be advisable, if the symptoms became urgent. A blister has sometimes appeared to me useful. Paracentesis has been performed, with temporary relief; but unless the primary disease be removed, of course the fluid will be reproduced. I should scarcely like to adopt the suggestion of Laennec, and inject slightly irritant fluids into the pericardium, so as to excite an inflammation that might, by causing adhesion, prevent the recurrence of the disease.

Œdema of the heart's substance is a mere anatomical curiosity.

Pneumo-pericardium and *hydro-pneumo-pericardium* are *affections of the extremest rarity.* I am not, indeed, aware of the existence of a positive example of the former. The fluid of pericarditis, fœtid and decomposed, has been known to furnish gas during life; so that the heart's action was accompanied with a "sound like that of a water-wheel"—(Bricheteau). I heard no such sound in the peculiar case already referred to, where the pericardium was perforated from the œsophagus (p. 183); but I satisfied myself of the presence of air in the sac by the change in position of tympanitic resonance, when the patient was turned from one side to the other.

ATROPHY.

Valvular Atrophy.—When the cordæ tendineæ of the mitral valve are shortened and extremely thin, they are, probably, purely atrophous. The larger tongue of this valve is sometimes purely defective in size, without obvious puckering, or other evidence of past inflammation. In both cases, regurgitation may occur. Reticulation is rare here.

The sigmoid valves may be thin and papery,—whence a sharp clicking state of the second sound, but no actual disturbance of the heart's action. These valves, too, may be reticulated or cribriform,* a state conceivably the cause of regurgitant basic murmur. But I have never known a murmur actually so produced; and if the thing were usual, the murmur signifying pulmonary regurgitation ought, instead of being one of the *mirabilia* of clinical practice, to be common,—seeing that reticulation is very closely as frequent in the pulmonary, as in the aortic, valves. Besides, there is an anatomical cause why, unless in extreme cases, reticulation should have no influence on the circulation; it affects those parts of the valves, close to their free edges, that lie surface to surface in the centre of the vessel, at the moment of its systole;—these particular portions of the valves have nothing to do directly with the prevention of regurgitation.

Heart.—(*a.*) The heart is said to be the subject of *concentric*

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* I speak of this state under the head of atrophy, to avoid multiplying divisions: it is very doubtful whether it is atrophous; neither do I think Bizot's theory (of rupture by extension from rapid growth) at all satisfactory. —(Mém. de la Soc. Méd. d'Obs. de Paris, tom. i., p. 367.)

atrophy, when the size and weight of the organ and the capacity of its cavities are alike diminished. In various degrees this form of atrophy occurs in wasting diseases, cancer especially; it appears sometimes to follow the tight embrace of pericardial false-membrane; to depend occasionally on narrowing and calcification of the coronary arteries; and, it is alleged (Laennec), has sometimes been artificially produced by the treatment for hypertrophy,—a statement which requires corroboration.

No local symptoms are positively traceable to this state; palpitation has sometimes been observed, but (as in advanced carcinoma) is, probably, rather the result of spanæmia, than of the atrophy. The impulse is deficient in force and extent,—the area of percussion-dulness lessened; of the character of the sounds, I know nothing of any importance. The pulse is small.

(*b.*) *Eccentric Atrophy.*—When the walls of the heart are greatly attenuated with or without subsidiary dilatation, loss of mass has occurred, and the state may fairly be termed one of eccentric atrophy. It is singularly rare; and is rarer still in the left than the right ventricle. In the latter situation it intensifies the effects of the dilatation and tricuspid insufficiency, with which it is commonly associated.

HYPERTROPHY.

§ I. *Valvular Hypertrophy.*—Thickening of the valves is sometimes observed in connection with hypertrophy of the left ventricle, when no anatomical or clinical indications exist of bygone inflammation; and where, in all probability, the thickening results from extra-nutrition consequent on the extra work entailed on the valves by the muscular hypertrophy. This state of the valves gives a dull, heavy, clanging character to the valvular portion of the first, and especially, to the second, sound; but is rarely carried far enough to produce either obstructive or regurgitant murmur.

§ II. *Cardiac Hypertrophy.*—Hypertrophy of the muscular substance of the heart may be *simple* (the affected cavity and its walls retaining their natural relative proportions), or *eccentric* (with dilatation of the cavities), or *concentric* (with contraction of the cavities). Hypertrophy may be general, in which case it is almost invariably eccentric; or, limited to a single compartment of the heart,—under which circumstances, the species varies. The left ventricle is the most frequent seat by far of the disease; next comes the right ventricle; then the left, and lastly the right, auricle. The signs and effects of hypertrophy of the different compartments of the heart differ so materially, that, to avoid confusion, it will be advisable to consider it in each situation separately.

I. *Hypertrophy of the Left Ventricle* (either pure, or combined with such slight dilatation or contraction as not to affect its own characters). First, of the physical signs,—taking a highly-marked case as the model.

(*a.*) Inspection discloses arching of the præcordial region, (especially in long-standing cases, and in early youth) with widening, but without bulging, of the left interspaces, from the third to the seventh. The impulse, increased in extent, especially to the left of the sternum, presents its maximum amount below and about the left nipple, and between this and the sternum; in character it is slow, heaving, and suggestive of pressure forwards steadily against an obstacle; in rhythm regular (unless there be some added morbid state); in force unequal. The amount of force may be sufficient to shake the head of the observer, the trunk of the patient, or the bed even on which he lies; such extreme power of action is rare, unless dilatation be combined with great hypertrophy. Of double systolic and diastolic impulse, I have already spoken (p. 172). The point of the apex-beat is carried downwards and outwards,—and may reach the seventh interspace (this is rare, however, without dilatation), at some distance outside a line let fall perpendicularly from the nipple. In eccentric hypertrophy the extent of visible impulse is much greater; the apex-point may be carried to the seventh space or eighth rib; the impulse may, without much difficulty, be felt in the back; its character is less heaving than in the pure disease, sharper, more knocking, or slapping, and the surface, over which it is perceptible to the hand, proportionally more extensive.

The superficial and deep-seated dulnesses of the heart are both augmented in area, and, probably, in amount also,—the parietal resistance is sometimes very notably increased. In dilated hypertrophy, the dulness may reach from the second interspace (here some slight increase upwards has occurred) to the eighth rib, and from an inch and a half to the right of the sternum to three inches and even upwards outside the vertical line of the nipple; and dulness may be detected in the back to the left of the spine. The rudely triangular form, natural to the heart's *superficial* dulness (*vide* Diagram II.), gives place to a dulness of somewhat square outline; and the deep-seated dulness is also more right-angled than in health;—this latter character, however, is not often to be satisfactorily ascertained.

In simple hypertrophy, the first sound is dull, muffled, prolonged, weakened in some cases almost to actual extinction, directly over the ventricle, the sensation reaching the observer's ear being rather one of impulsive motion than of sound;—under these circumstances a tolerably full systolic sound may, nevertheless, frequently be found at the base and at the ensiform cartilage; the extent of its transmission is very limited; the second sound, though wanting in clearness, may be full and clanging; the post-systolic silence is shortened. During palpitation the first sound sometimes becomes comparatively clear. In hypertrophy with dilatation, the sounds gain greatly in loudness, and extent of transmission, especially if the valves be perfectly healthy and free even from hypertrophous thickening; and the tone of the first at the left apex is notably clearer than natural. Reduplication of either sound, sometimes occurring, possesses no special character: it is not common.

Systolic blowing murmur, basic and audible at the second right cartilage, is sometimes heard in cases of pure hypertrophy; nor can it be positively ascribed in all instances to coexistent spanæmia. I have known such a murmur disappear, when the heart had become comparatively quiescent by treatment of a depressing kind rather than otherwise, and wholly non-ferruginous. Hence excess of force of propulsion of naturally constituted blood would seem capable of generating *direct* murmur. Hypertrophy may possibly, during the excitement of palpitation, induce mitral regurgitant murmur by disturbing the action of the papillary muscles. Such murmur actually does, as a clinical fact, exist at one time and disappear at others. And systolic basic murmur may also, very probably, be generated in cases of dilated hypertrophy, in consequence of the altered relationship of the aortic orifice to the cavity of the ventricle—altered both in point of size and of direction of the blood current (pp. 216, 220). In dilated hypertrophy, knocking and rubbing additions to the first sound at the apex, either left or right, are not very uncommon. Possibly, too, the second sound may be intensified at the left apex by the abrupt recedence of the enlarged heart from the side during its diastole.

The respiration at the centre of the cardiac region (upper sterno-costal angle of the fourth left interspace) is feeble and distant,—but not so feeble nor so distant as it would be with an equal amount of percussion-dulness from fluid in the pericardium.

In estimating the dulness really depending on an hypertrophous heart, the observer must bear in mind, that its apparent extent may be, on the one hand, increased by engorgement of the right cavities, from temporary disturbance of the circulation, by aneurism of the aorta, by indurations in the lungs, pleura, or mediastina, by tumors of the œsophagus even, and by enlargements of the liver, which, by pushing the organ upwards and to the left, increase the area of dulness in those directions;—and, on the other hand, decreased by emphysema and bronchitic distension of the lung.

(*b*.) The state of the functions may be described as follows, in cases of pure hypertrophy, or hypertrophy without any great excess of dilatation. The strength does not seriously suffer, unless the disease be carried to a great height; the power of walking and of ascending hilly ground is diminished, not from feebleness, but from the dyspnœa and oppression induced by the attempt. Patients generally lie with the head high: the colour of the integuments varies; if the hypertrophy be pure, the face is florid, if coupled with moderate dilatation, there may be slight purpleness and lividity; but marked purple discolouration does not occur, unless there be very considerable dilatation, valvular obstruction, or pulmonary disease. Hypertrophy of the left ventricle does not *per se* produce œdema of the ankles, much less general anasarca: even hypertrophy and dilatation, unless the latter be in great excess, fails to induce this evidence of systemic vascular obstruction. Hope, as is well known, maintained the reverse, holding that pure hypertrophy, if protracted, will produce general dropsy; but he gives no cases demonstrating the fact, and the motives of his belief are, as far as he shows, totally speculative. And *à priori* views are not so completely in his favour as he appears to imagine; when he talks of the "increased force of circulation surmounting the natural tonic power of the capillaries," he forgets that that very tonic power may have increased *pari passu* with the growth of the hypertrophy. The question is one of observation; and I have stated what has fallen under my own notice.—The muscles are well nourished and of good colour. There is not any form of dyspeptic derangement particularly assignable to hypertrophy; the disease may exist for years without materially affecting the digestive powers, provided moderate exercise be taken. Constipation acts as a source of habitual annoyance. There is more or less dyspnœa, either constant or occasional,—in the latter case induced by the most trifling effort: the pulse and respiration-ratio may be perverted in consequence; paroxysms of dyspnœa have not occurred under my observation, unless there were much dilatation, valvular obstruction, or pulmonary disease. Dry cough annoys some patients; I have not observed œdema of the lungs;—the radial pulse, in no wise peculiar in regard of frequency, and perfectly regular in rhythm, is full, strong, firm, tense, resisting, and prolonged, without jerk or thrill, in the pure disease; if dilatation be superadded, it retains its fulness, but loses in some measure strength and resisting power. It is said not to be increased in frequency, as in health, by change from recumbency to the sitting and standing postures. The action of the carotids is visible; and various throbbing sensations are felt in the head. Præcordial pain, rare in simple hypertrophy, is not uncommon in the dilated variety, ranging in severity from a slight aching sensation to the severe suffering of pseudo-angina. Paroxysms of such pain may be accompanied with, and probably depend sometimes on, congestion of the lungs and loading of the right cavities of the heart with blood; in some instances they are distinctly traceable to intercostal neuralgia. The common action of an hypertrophous left ventricle would be palpitation (if not in frequency, in force), to a healthy person; under excitement, or often without apparent cause, a violent fit of throbbing action comes on—regular, however, or almost so, in rhythm, producing violent pulsation in the neck and head, with tinnitus aurium. Cephalalgia, dull, aching, or throbbing, is of more frequent occurrence than in healthy persons, but by no means a constant symptom; sensations of rushing of blood to the head are common, especially on stooping, and, indeed, on sudden movement of any kind. The intellect is habitually unaffected, as regards any symptomatic state clinically significant; no proof exists of its being brightened; nor, on the other hand, unless towards the close of life, have I found pure hypertrophy render individuals incapable of ordinary mental exertion. Concerning the influence of hypertrophy in producing cerebral hæmorrhage or acute encephalitis, recorded facts do not permit a positive affirmation. The favourite *à priori* physiological doctrine, that apoplexy is a sort of appanage of the heart affection, is at the present time not only non-demonstrated, but actually controverted by attainable evidence, so far as this goes. Still, the character of that evidence is imperfect, as I think I have elsewhere succeeded in showing;* and the whole subject calls for fresh investigation. It may eventually be found that the fatty diathesis is the real link between cerebral hæmorrhage and heart disease,—fatty enlargement of the heart on the one hand, fatty degeneration of the cerebral vessels on the other. Reflex phenomena, at the moment the patient drops off to sleep, for example, sudden starting of the legs, are not very uncommon; they may possibly be traceable to active congestion of the spinal chord. The eyes of some patients are bright, full, prominent, prone to injection; and by such persons visual illusions, luminous vision, and muscæ volitantes are frequently complained of. Epistaxis seems to be more usual than in individuals of equal age free from hypertrophy.

Hypertrophy of the left ventricle is said to entail increase, in weight and substance, of the organs generally; and to enlarge the calibre, thicken the coats, and even increase the length of the arteries: but the age at which hypertrophy of the heart becomes common, is precisely that at which a natural tendency to enlargement of the vessels,—if indications given by the statistics of M. Bizot may be finally accepted,—is devoloped. The alleged influence of hypertrophy in producing renal disease with albuminuria, is not established: there may be some connection, through the fatty diathesis, between pseudo-hypertrophy and the fatty alteration of the kidney. I have not known pure hypertrophy produce albuminous impregnation of the urine.

(*c.*) Simple hypertrophy, of medium amount, by means of regulated diet, moderate exercise and general attention to hygienic rules, may be rendered a very endurable affection. If it be of considerable amount, and the patient, instead of living according to rule, is forced to work laboriously, and live irregularly, the probable issue is death, through secondary affections, complications and functional derangements, originally unconnected with the heart-affection, rendered more serious by its existence. But hypertrophy alone is rarely, and I do not think ever rapidly, the direct cause of death: I cannot call to mind any case, where I have actually known it, and it alone, positively fatal. Hope, it is true, affirms he has known hypertrophy destroy life in some instances within a year of its commencement (Op. cit. p. 278); still he cites no cases, and appears from the context to have in his mind's eye examples of dilated hypertrophy variously and seriously complicated. Dr. Latham is of opinion, "the heart, *by the simple vehemence of its action*, has the power to kill,"—through cephalalgia, insomnia, delirium, mania, convulsion, and nervous exhaustion (Diseases of the Heart, vol. ii., p. 338). But by the phrase, "simple vehemence of action," we are not to understand (however this may appear warranted by its terms) such action as an unaided hypertrophous heart in its highest degree can engender. For Dr. Latham gives no proof that the organ possesses any such power, and the only positive case referred to in illustration of the above opinion, is one of hypertrophy and dilatation of the left ventricle following endo-pericarditis and bygone dropsies;—where, too, (no *post-mortem* examination took place) there *may* have been disease within the cranium.

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* Clin. Lect., loc. cit., p. 279.

The more dilatation predominates over hypertrophy, the more serious becomes the prognosis.

(*d.*) With what hopes may the *treatment* of pure, or somewhat dilated, hypertrophy of the left ventricle be undertaken? Is the disease curable? Can the nourishment of the heart be not only controlled by artificial means, but reduced below the standard of health? Drs. Latham, Taylor, and Blackiston emphatically deny that art is possessed of any such power; Laennec and Hope maintain that the feat is easy of accomplishment. But Hope repudiates the plan recommended by Laennec, and the simple common-sense system lauded by himself fails utterly in effecting the textural changes he ascribes to it. For my own part, I have never known the cure of indubitable hypertrophy proved by physical signs, and hold it unwise to promise any such result from treatment.* But it is not difficult to remove or greatly mitigate the symptoms of simple hypertrophy in the majority of cases, and render life not merely tolerable, but comfortable.

The theroetical indication is very obviously to tranquillise the heart by diminishing the quantity, without deteriorating the quality, at least materially, of the circulating fluid. For this purpose, occasional bleedings from the arm, to the extent of four, six, or eight ounces at a time, at intervals of from two to six weeks, are recommended by Hope, in conjunction with a diet mainly consisting of farinacea and vegetables. But even loss of blood to the extent here intended, is more than can be borne by the majority of persons, without slight impoverishment of the fluid; and the least amount of anæmia deeply aggravates the dangers of hypertrophy. Besides, it has appeared to me that over-action of the heart is quite as effectually and as lastingly controlled by very moderate cupping or leeching over the præcordial region, as by the abstraction of a comparatively large quantity of blood from the arm. Four or five leeches, even, will sometimes calm the excitement of a powerful left ventricle, in a state even of somewhat dilated hypertrophy,—and this in a well-grown adult.

There is no known drug possessing the faculty of directly controlling the growth of the heart; iodine seems quite valueless: general principles alone guide us in attempting to put a term to the increase of its bulk. Purgative medicines, and I believe the saline and aloetic the most appropriate of the class, aid the good effects of local bleeding. Diuretics are useful, quite independently of the existence of dropsy. Direct sedatives of the heart, hydrocyanic acid, acetate of lead, digitalis, and belladonna (the latter both internally, and in the form of plaster to the surface) must be employed, with occasional intermissions, during the entire treatment of the case. Of all medicines of this class aconite seems the best; the alcoholic extract of the root may be given in doses of one eighth of a grain with perfect safety: no drug, that I know of, possesses so fully the power of relieving painful sensations and disquietude about the heart.

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* Even the physical signs may, unless he be very cautious, betray the observer into error in regard of this matter. Thus, not only the impulse may be reduced in force and extent, and the character of the first sound changed, but the area of percussion-dulness lessened (by disgorgement of the right cavities), and yet the heart's actual mass remain precisely as before. All this may sometimes be done, by treatment, in a few days; sometimes not in months.

If there be anæmic tendency, animal food should be allowed, —under all circumstances, indeed, fish may be permitted. Alcoholic fluids, of all kinds, must be avoided; and fluid taken, as a rule, in but small quantity. Moderate exercise is advisable.

In treating a case of this kind, the patience of the physician must never fail him: it may require months, nay, years, to produce a favourable effect on the disease,—and want of steadiness of purpose and conviction may, in a few days, undo the good accomplished by the efforts of previous weeks. There is one caution to be given to the young practitioner,—that he never push the treatment to the extent of producing anæmia; the superaddition of anæmia to hypertrophy is that which gives a really ominous character to the latter.

II. *Concentric Hypertrophy of the Left Ventricle.*—The discovery that the contraction of the ventricle occurring *in articulo mortis* might give to a heart simply hypertrophous the appearance of one concentrically hypertrophous, led, for a time, to the almost complete rejection of concentric hypertrophy as a possible state. But its occasional, though rare, existence is matter of absolute fact. The symptoms and signs are those of simple hypertrophy; theoretically the disturbance of the circulation will be greater, but clinical illustrations of the point are wanting.

III. *Hypertrophy of the Right Ventricle* is comparatively rare in all forms, and excessively rare unless associated with dilatation. The signs of this combination are arching of the lower part of the sternum, with greater or less eversion of the ensiform cartilage, and fulness of the epigastrium. The left costal cartilages may be more bulged than the right,—a circumstance accordant with the fact, that though the impulse plays very forcibly against the sternum and ensiform cartilage, it inclines, in cases of highly marked right hypertrophy, to direct itself, in consequence of slight displacement of the entire organ, more against the edge of the left, than the right mammary region. Hence it is, that the form of the præcordial region and the site of impulse might betray the observer into the notion that the left, and not the right, ventricle was the seat of hypertrophy. The percussion-dulness may extend considerably beyond the right edge of the sternum; I have known it reach an inch outside that edge in a child aged ten years. At the same time the dulness may be carried unduly to the left also. The distended and enlarged right auricle is the source of much of the percussion-dulness to the right of the sternum.

Dilated hypertrophy of the right ventricle widens the tricuspid orifice in the majority of cases; unless the valve grow in proportion to the widening of the orifice, a regurgitant murmur should, theoretically, occur. Yet, such murmur certainly does not occur by any means constantly. Can this be explained by some constrictive action of the orifice during life preventing regurgitation? The first sound is duller than natural at the ensiform cartilage; the second fuller, stronger, and more accentuated. Visible jugular pulsation sometimes exists, without the tricuspid valve being incompetent.

Marked hypertrophy of the right, is so rare without hypertrophy of the left, ventricle, or some form of valvular disease, that it is difficult to give a really clinical transcript of its symptoms. Lividity of the face and subcutaneous œdema about the face and neck sometimes exist. Theoretically the lungs must suffer,—and hence dyspnœa, engorgement and œdema of the pulmonary parenchyma, bronchitis, pneumonia, tubercles, pulmonary apoplexy, and hæmoptysis, have all been set down by various systematic writers, as dependencies of the affection. Now, of all these alleged symptoms, dyspnœa is the only condition that seems to have thoroughly made out its claim to be so entitled. I believe, however, that engorgement and œdema of the tissue of the lung is not uncommon. Pneumonia, pulmonary apoplexy, and hæmoptysis, I have certainly seen in cases of right hypertrophy; but in these instances, mitral regurgitation existed also. The notion that right hypertrophy tends to tuberculise the lungs, is not supported by anything I have observed.

The radial pulse, from its being free from all peculiarity of character, affords valuable aid in the diagnosis. Natural in force, while the cardiac action is strong, its state argues against the existence of hypertrophy of the left ventricle, to which some of the local physical signs might otherwise point. The pulmonary artery, of course, bears the force of the hypertrophous ventricle; a fact probably explanatory of the frequency with which, under these circumstances, its coats are atheromatous.

The difficulty of circulation through the vena cava and jugulars tends to congest the brain venously.

IV. Hypertrophy of the left auricle often accompanies, in variable degrees, constrictive and regurgitant disease of the mitral orifice; dilated hypertrophy of the right auricle, with chronic thickening of its endocardium, I have seen carried to a great extent in cases of dilatation of the tricuspid orifice. The symptoms of the diseased state of orifice, whatever they are, are intensified by these auricular hypertrophies; of signs peculiar to them, there are few (*vide* p. 210).

DILATATION.

I. By dilatation of the heart, is understood that state in which the capacity of its cavities is increased disproportionately to the thickness of their walls. It occurs in three forms: *hypertrophous dilatation,* where dilatation predominates, but the walls are somewhat thicker than in health; *simple dilatation,* where the walls are of such thickness as would be normal, had the capacity remained unchanged; and *attenuated dilatation,* in which the walls are distinctly thinner than in health,—to such a degree that the thickness of the wall of the left ventricle may, in some points, positively not exceed one line. Clinically, as well as anatomically, the characters of dilatation are obvious in proportion to the excess of capacity over thickness; and the following description must be understood to refer to cases in which such excess is prominently marked. Dilatation of this kind most frequently affects both ventricles; one, however, to a greater amount than the other: it is not uncommonly limited to the right, very rarely to the left, ventricle. The tissue of the dilated walls is more or less soft and flabby—and if this deficiency of firmness be disproportionately marked at any particular spot, the ventricle may there yield, undergo pouching, and a true aneurism of the heart ensue. The form of the dilated ventricle becomes globular, and the apex of the heart so rounded off, that it may with difficulty be detected. The transverse measurement of the organ undergoes much greater increase than the vertical.

II. Dilatation was formerly regarded as the condition of the heart mainly inducing the important class of dropsical symptoms, depending on passive and mechanical systemic congestions. This opinion, discountenanced by Bouillaud, under the apprehension that valvular obstruction was their sole cause, was restored to favour by Hope and Andral,—the former of whom, indeed, as we have seen, went the length, on probably insufficient grounds, of teaching that pure hypertrophy, also, was capable of generating dropsy. Of late, Dr. Blackiston has brought together a body of evidence calculated to show that the systemic vessels do not become loaded in cases of dilatation, unless there be co-existent tricuspid regurgitation. Still more recently Dr. H. Douglas has defended the original thesis of our forefathers. It becomes necessary for me here to express an opinion on this "vexed question;" and, in order to avoid the necessity of again returning to the subject, I will throw into a series of propositions such inferences, as seem admissible, bearing on the general doctrine of the production of dropsy by diseases of the heart.

1. Mitral regurgitation or obstruction, or aortic regurgitation or obstruction, may severally exist, and, for a lengthened period, without systemic dropsy supervening. 2. This proposition continues true, whether hypertrophy exist behind the obstruction or not. 3. Simple hypertrophy of the left ventricle may reach the highest point without systemic congestive effects of any kind. 4. Dilated hypertrophy, even, of the left ventricle, may last for years without any such effect, provided the dilatation be not in notable excess. 5. The heart may be weak and feeble, or actually in a state of fatty disorganisation, and the pulse feeble and irregular, and yet no systemic congestions occur. 6. The natural relationship of width of the arterial orifices, and also of the auriculo-ventricular orifices, may be materially perverted, without the least systemic dropsy arising.* 7. Tricuspid regurgitation, where the right ventricle is in a state of dilated hypertrophy, as shown during life, by swollen and pulsatile jugular veins which fill from below, and as shown after death by actual examination, does not necessarily produce dropsy.† 8. It would appear, then, that something beyond all these cardiac

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* Case of Cyanosis, Med. Chir. Trans., vol. xxv., p. 1.

† W. Hallington, ætat. 53, died at U. C. H., April 7, 1849, with fatty dilated enlargement of the heart (weight 17½ oz.), dilatation of the tricuspid and mitral orifices, and aortic regurgitation carried to a great extent, and yet not a particle even of œdema about the ankles had appeared. The patient had also an aneurism of the aorta, at its bifurcation; but this could not be held to be *preventive* of dropsy.

conditions is wanting to ensure the occurrence of dropsy, unless on the gratuitous assumption that, were life sufficiently prolonged, they would in themselves of necessity induce it. What that something beyond the heart, conducive to dropsy, probably is, I will by-and-bye state. 9. The cardiac affections most frequently connected, as matter of experience, with systemic dropsy, are dilatation and tricuspid regurgitation; and it is certainly very rare for either of these states to exist for any length of time without the supervention of such dropsy: any hypothesis, explanatory of cardiac dropsy, must look to these states as forming important links in its chain of causes. 10. But the something beyond the heart? This is probably furnished by certain conditions, favourable to transudation of the serosity of the blood, in that fluid itself, in the walls of the capillaries and venous radicles, and in the receiving tissues. As concerns the blood, the influence of an impoverished state of that fluid is too well known to be for a moment contestable. Again, it is readily conceivable that the variable density of texture of the walls of the vessels shall promote or restrain the process of filtration. Lastly, cases occasionally present themselves, in which dropsy, supervening from diseased heart, fails to affect portions of the body, noted, under ordinary circumstances, as the earliest and readiest sufferers,—for instance, the lower extremities. I have observed this where the legs had been the seat of erysipelas and subcutaneous inflammation prior to the occurrence of the cardiac dropsy: the chronic anatomical changes in the cellular tissue in such a case possibly act as a barrier to its reception of serosity from the vessels. Dilatation of the heart, occurring as a primitive disease through simple weakness, or actual organic alteration of the texture of the organ, may be easily supposed *à priori* capable of generating systemic congestion and its results. In a heart so affected, the necessary *vis à tergo* is deficient,—capillary stagnation ensues. Now this very stagnation, becoming habitual, may modify the qualities of the blood; and impair the nutrition of the walls of the vessels through the strain they suffer. But change in the blood is, in all probability, worked out meanwhile by other and more effectual agencies.

III. The physical signs of general dilatation may be set down somewhat as follows:—The apex-beat is very indistinctly visible, or, if the patient be full in person, actually invisible; when seen, it may fall *within* the natural point of the apex-beat, in consequence of the rounded-off form of the heart destroying the true apex; if the dilatation be considerable, the apex strikes the surface *below* the fifth interspace. The impulse, whether more extended than natural, or limited to the fourth and fifth interspaces, frequently exhibits the quasi-undulatory character. There is no prominence of the cardiac region. As felt by the hand, the impulse is feeble in proportion to the purity of the dilatation: the apex-beat, though visible, frequently cannot be distinguished by the finger from the impulse of the ventricles generally: the impulse, non-vibratile, either consists of a short feeble slap, followed by a sudden fall back of the organ, or of a more prolonged faint tremulous motion: the force of successive beats is unequal; their rhythm may be irregular to a slight or to the very highest degree: there is a want of perfect uniformity in the point of the surface struck by the hand in the successive beats of a series, quite independently of the influence of respiration: palpitation may notably increase the force of impulse, though it often does so but very slightly. The intensity of percussion-dulness in the superficial cardial region is not, as it probably is in hypertrophy, increased; and in cases of attenuated dilatation the parietal resistance may be less marked even than in the state of health. The areas of dulness, deep and superficial, are both widened; the former inclines to squareness of outline, the transverse diameter of the heart having proportionally undergone a notably greater increase than the vertical. The systolic sound short, abrupt, and unnaturally clear, both at the apex and base, appears near the surface; its maximum point is generally slightly lowered. The diastolic successor is not specially affected. Both sounds, as heard at the top of the sternum, bear the natural relationship, in point of intensity and clearness, to those audible over the heart itself. If the dilated ventricle be soft, flabby, or fatty, the first sound may be very weak, faint, and toneless; and the second so feeble as to be inaudible at the apex, especially if the heart's rhythm be markedly irregular. Upon the quality and mass of the heart's texture will depend the extent to which the sounds are transmitted through and over the chest; if the dilated ventricles be well nourished, and *à fortiori* at all hypertrophous, the sounds (approaching those of hypertrophy with dilatation) will be widely diffused, and clearly audible in the right back; if, on the contrary, the dilated heart be a soft, flabby one, the area over which its sounds are audible may be extremely limited. A slapping sound, the intensity of which may be increased by causing the patient to bend forwards, is sometimes produced by the collision of the heart against the side. Dilatation is not productive of any intra-cardiac murmur, unless the morbid state has proceeded to such lengths as to render certain valves incompetent to close their orifices: this may occur with the tricuspid, more rarely with the mitral, and still more rarely with the aortic valves. The murmurs thus engendered are, of course, always regurgitant; but they do not always occur when their physical cause, incompetency of the valves, actually exists, inasmuch as the strength of the ventricles is sometimes incapable of either giving the necessary impetus to the regurgitating stream, or of calling forth such energy of its systolic reaction on the part of the aorta, as is required for the production of murmur. Are reduplications common when the ventricles are unequally dilated? I think so; but my number of observations on the point is too small to justify a positive assertion.

Dilatation of the right ventricle in particular is signified by excess of epigastric pulsation, by jugular turgescence, and, if there be either tricuspid regurgitation or some muscular strength in the wall of the ventricle, by jugular pulsation.

Direct evidence of dilatation of either auricle is only to be had by percussion in the natural sites of those cavities: when the jugular veins are permanently dilated and knotty, the existence of dilatation of the right auricle is inferrible almost as matter of necessity.

IV. Taking, as our imaginary model, a case of highly marked general dilatation of the heart, the failures of the various functions will be found much of the following kind:—(*a.*) The patient, habitually irritable and melancholy, breaks from time to time into fits of despondency and petulant complaint; he is deficient in energy, both mental and bodily—the dislike to movement is only overcome under the persuasion that exercise is in some measure essential; the strength fails; the weight, unless factitiously sustained by dropsies, falls very notably; sleep, in the true sense of the term, is rarely enjoyed,—fitful dozes, abruptly interrupted by startings and frightful dreams (incubus is not more common than in persons free from heart-disease), are its nearest substitute; habitually the patient lies with his head high, and when under the pressure of a fit of dyspnœa (cardiac asthma) he bends the head forwards, or sits erect with the feet hanging out of bed; his debility and dropsical unwieldiness prevent him from assuming some of the peculiar postures in which the subject of *pulmonary* spasmodic asthma struggles for breath. (*b.*) Chilliness of the extremities, livid discolouration of the prominent parts of the face, mottled with patches of sallow and earthy tint, or varied by leaden or almost black discolouration about the eyes and mouth (conditions of colour all of them most marked, as a rule, in the morning); lividity or blueness of the lower extremities generally, with excess in particular spots,—spots which, eventually falling into a state of absolutely stagnating circulation, slough, independently of calcification or inflammation of the arteries or veins; and, lastly, anasarca, softly pitting, spreading from the feet to the abdominal, and even the thoracic, walls, the external genital organs, the face and neck, rarely the upper extremities: all these conditions show how deeply the tegumentary system suffers. (*c.*) The joints remain unaffected. (*d.*) The digestive organs are less fortunate. The tongue is broad, full, not always pitted at the edges, however, and of dark purplish ground,—the fauces venously injected; hæmorrhage may occur from various parts of the alimentary mucous tract,—I have seen this in the case of the bowels, when the dilatation alone seemed its direct cause, but do not feel positive that hæmorrhage from the stomach has occurred under my observation, unless there were intermediate high congestion of the liver (the hepatic system may act as a sort of safety-valve for the stomach, until itself grows overloaded). Hæmorrhoids are not so common as might be expected,—the bowels habitually constipated, or alternately relaxed and confined,—the discharges dark. The liver is more or less congested: the hepatic system suffers first, the portal secondly; unless both are implicated, the size of the organ is not sufficiently increased to affect the results of percussion notably: the texture of the lobules may remain perfectly sound. Positive jaundice does not occur from this state of the liver alone; but I have seen a faint yellowish tint during life, where death disclosed nothing but hepatic congestion to account for it. Ascites follows on (I have no experience of it as a precursor of) anasarca. (*e.*) Venous stagnation, with its consequences, occurs in the lungs,—oppressed breathing, amounting even to orthopnœa, complete and habitual, with occasional asthmatic paroxysms, and cough, dry, harassing, and even convulsive, or accompanied with expectoration, serous, rarely frothy, or stained or streaked with blood, or mixed with a little blood, fluid and dark, or in pellets. The physical signs of bronchitis, or of irregular pulmonary congestion (most marked, however, at the posterior bases), of pulmonary œdema, or of pulmonary apoplexy, may be found,—actual hæmoptysis to some amount may accompany the latter state. (*f.*) Feeble, fluttering, distressing palpitation, increased by the slightest movement, occurring from some obvious cause, or as frequently without apparent excitement, is a standing source of misery to those sufferers. So, too, is uneasiness in the cardiac region, of characters most difficult to describe,—of an intensity varying between a mere sensation which constantly reminds patients that (as they often express themselves) "they have a heart," and the agony of angina,—paroxysms of which may actually occur and put an end to existence. Tenderness on pressure may be discovered over the heart in some cases. The pulse is either small and feeble, and abnormally posterior in time to the ventricular systole, but regular; or it is narrow, feeble, fluttering, and irregular. The latter state is either limited to the periods of palpitation, or, if constant, is indicative generally of softening of the heart. True intermittence is rare. The superficial pulses are not visible. Faintness, occasionally lapsing into actual syncope, occurs from time to time. (*g.*) Swelling of the abdominal lymphatic glands is said sometimes to occur: I have never seen it. (*h.*) The kidneys are passively and mechanically congested like the lungs and liver; the renal regions may be tender on deep pressure posteriorly; but I have never known the organs sufficiently increased in bulk to cause any positive extension of the area of their posterior dulness. The urine is small in quantity, high coloured, loaded or not with lithates; albuminuria may occur from mere renal congestion,—the urine continues of good specific gravity, and exhales no whey-like odour. The symptoms often disappear spontaneously, and may almost always be quickly removed by certain measures directed to the kidneys, especially cupping or dry-cupping. (*i.*) Sexual inclination is weakened materially; rupture of a dilated heart has, however, occurred *in actu coitûs.* It is said that uterine hæmorrhage is sometimes traceable to dilatation of the heart: the statement is not confirmed by any cases that have fallen under my notice, and seems to stand in need of corroboration. (*k.*) More or less congestion of the encephalon can scarcely be escaped, where the face exhibits marked indications of that state. The dull cephalalgia, inability to exercise the mind, heaviness, torpor, somnolence, semi-coma, and finally, coma, that mark the progress of these cases, are thus explicable. Cerebral hæmorrhage and acute softening stand in undetermined relationship to dilatation. (*l.*) Congestion of the cord may be the cause of certain reflex phenomena, convulsive cough, startings from slumber with affright, and convulsive actions of short duration on waking from sleep. (*m.*) Muscæ volitantes, luminous vision, fulness, wateriness, and injection of the eyeballs; tinnitus aurium, dulness of hearing, and deficiency of smell, and occasional epistaxis, from congestion of the Schneiderian membrane; all indicate participation on the part of the organs of sense.

Taking the systemic and the pulmonary obstructions as two separate classes, the former have occurred under my observation almost invariably before the latter: Hope taught the converse, but it appears to me on theoretical grounds.

It is difficult to affix specially to dilatation of the right ventricle any symptoms that may peculiarly depend upon it,—so frequently does some amount of dilatation of the left ventricle or some form of valvular impediment exist to complicate the problem. Theoretically, the signs of deficient oxygenation of the blood will be marked in proportion to the amount of pure or attenuated dilatation of this ventricle,—as will, also, the congestive influences on the brain and abdominal viscera.

V. The *diagnosis* of dilatation of the heart turns mainly on the following points:—Weak action, quasi-undulatory impulse, indistinctness of the apex-beat; increased area, with squareness of outline, of the percussion-dulness,—the whole not lowered in proportion to its width; clearness, shortness, abruptness of the systolic sound, or great deficiency of tone in certain cases, with prolongation of the post-systolic silence; peculiar characters of the pulse; and signs and symptoms of systemic and pulmonary obstruction and congestion.

Simple hypertrophy is distinguished from dilatation by the forcible action of the heart; by the præcordial bulging; by its distinctly localised thrusting impulse; by the heart being lowered in proportion to the general area of dulness; by the muffled dulness and prolongation of the first sound and shortness of the first silence; by the characters of the pulse; and by the systemic signs being those rather of an excited than of a stagnating circulation. Every one of these characters differs more or less from those of dilatation.—*Eccentric hypertrophy*, also, has its forcible action, præcordial arching, and distinct apex-beat; the heart is lowered in the proportion of its bulk; the systolic sound is loud and powerful; and the pulse differs from that of dilatation. The only difficulty comes of the fact that if the cavities be more increased in capacity than the walls in thickness, the systemic and pulmonary signs may incline to those of pure dilatation.—*Mitral regurgitant* disease will, under ordinary circumstances, be distinguished from dilatation by its special systolic murmur at the left apex, and by the more forcible impulse: for some amount of hypertrophy in the vast majority of cases ensues on the valvular disease. Still there are two kinds of possible fallacy: dilatation may be attended with dynamic mitral regurgitation; and, on the other hand, organic mitral regurgitation may, *pro tempore*, be murmurless. Now, the first difficulty will occur where the orifice is so much dilated that its valve, though perfectly sound, has ceased to be of sufficient size for purposes of complete closure. There is no possible means of distinguishing such regurgitation from that induced by actual disease of the valve; happily, the phenomenon is very rare; and, as the other characters of dilatation must be present in a high degree, the treatment will not err. The second difficulty—that of murmurless organic regurgitation—will arise from feebleness of the backward current, itself depending on temporary general collapse, or weakness limited to the heart; in a short while the heart's vigor having improved, mitral murmur becomes audible.—Dilatation differs from *chronic pericardial effusion* by the square outline of its dulness; by the distance from the clavicle to the spot of the apex-beat being natural or increased; by the sounds being superficial, and almost as feeble at the præcordial region as at the top of the sternum; by the dulness not extending above the third rib; and by the total absence of friction-sound, no matter in what position the patient be placed.

VI. Under all circumstances a most serious disease, the danger of dilatation increases directly as the excess of the capacity of the cavities over the thickness of their walls; directly, too, as the softness and flabbiness of the heart's tissue; directly, too, as the general deficiency of tone in the system and impoverishment of the blood. Once dropsy has supervened, life can with difficulty be prolonged by art beyond twelve or eighteen months.

Dilatation of indubitable existence is not removable by *treatment:* those doubtful cases of the disease, which Hope professed to cure, bear no claim to scientific recognition. But if it be not, in the highest sense, a curable affection—though art cannot remove dilatation, art can render dilatation bearable, and even *unfelt.* In conducting the treatment, the essential element of the disease, *weakness* (whether the disease be primary or secondary), must be constantly held in view, all debilitating measures systematically avoided, and the effort made to improve the heart's tone without exciting its irritability.

(*a.*) Unprovided, as we are, with any medicine exercising a specific action on the tone of the heart's fibre, general tonics must be resorted to. Bitters, mineral acids, and preparations of iron, in doses and combinations modified according to circumstances, supply the groundwork of medicinal treatment: it is of course to be understood that no positive contra-indication to the use of these agents exists beyond the heart. Belladonna, internally and externally, tranquillises excited action better and more safely than other sedatives. Aconite may be given in very small doses, on urgent occasions, but by no means employed habitually. The exhibition of digitalis requires the utmost caution: slackening the circulation, as it does, it promotes coagulation within the heart, or, in a less degree of its action, accumulation of blood in the cavities, whereby they may be still further passively dilated. If the power of the ventricles be seriously impaired, digitalis cannot be given without excessive risk, and had much better be altogether avoided. If the heart be nervously excited, the various anti-spasmodics are indicated. The inhalation of chloroform or æther cannot be permitted.

The condition of the chylopoietic viscera must be carefully watched,—due action from the bowels ensured daily, without weakening purgation (aloetic medicines are the best), and the action of the liver, if this be sluggish, promoted by taraxacum and an occasional mercurial aperient.

The patient should lead a tranquil life, avoiding all excitement, but mixing in cheerful society; his exercise should be moderate (always considerably within fatigue); gentle use of light dumb-bells, by promoting the circulation through the upper extremities, seems to act beneficially; but caution is requisite in permitting this. A good nourishing animal diet, of plain, easily digestible character, is that fittest for these patients. Much drink of any kind is to be avoided; bitter beer in moderation is allowable at dinner (or, if the patient have been accustomed to take these, a glass, or at most two, of port or sherry); dandelion coffee is a good material for breakfast. A dry, bracing air, as a rule, agrees best. Particular circumstances, it is true, may require the patient to live in one of the very opposite characters; but, though a relaxing atmosphere may relieve an accompanying dry bronchitis, it unquestionably tends to depress still further the heart's energy. Flannel next the skin is indispensable; the shower-bath may be cautiously tried, and continued or not according to its visible effects,—effects which, it has appeared to me, are, in all diseases, much under the influence of habit in respect of ablutions, and idiosyncrasy. Tepid salt-water bathing, or even, when the affection is not far advanced, quiet immersion in the open sea, is useful; swimming exercise, it is scarcely necessary to say, should be absolutely avoided.

(*b*.) Paroxysms of dyspnœa may be relieved by hydrocyanic acid, cannabis indica, æther, and liquor opii sedativus, and the æthereal tincture of lobelia inflata; by dry-cupping, or the application of three or four leeches to the præcordial region (especially if there be palpitation). If disturbed action be traceable to hysteria, the usual remedies for that state should be given; if to flatulence, carminatives sometimes tranquillise the organ almost instantly. Pulmonary congestion and sub-inflammation require dry-cupping, mustard poultices, flying blisters; and, as they are rarely active, squill and ammonia or senega internally. Should actual pulmonary inflammation occur, antimony must be employed with great caution: it is easier to depress the vital powers than to raise them. Systemic and serous dropsies are removable by the methods elsewhere described (*vide* Diseases of the Valves); they should not be too rapidly removed, supposing surgical influence becomes requisite, as the sudden loss of support on the part of the vessels may induce prostration of strength, from which the patient cannot be recovered.

CHANGES OF CONSISTENCE.

§ I. *Softening.*—I. In addition to cases in which softening of the heart results from acute inflammation, hæmorrhagic infiltration, or fatty disorganisation of its texture, that state is met with in certain acute febrile affections—for instance, typhoid and typhus fevers; and in some chronic blood diseases, as scurvy and purpura. In all these instances, softening constitutes an accident of the primary affection; but there are other cases in which the heart notably loses its consistence, without there being any prominent blood-disease, or any obvious affection of the organ itself, except the deficiency of firmness. To the latter cases alone it is proposed at present to refer.

II. The impulse of a heart of this kind may be invisible, or visible with some and invisible with other beats, and occasionally somewhat undulatory. If any single beat be of considerable force, the left ventricle is very certainly somewhat hypertrophous as well as soft. Percussion discloses nothing, unless, as is often the fact, the flaccid organ be dilated also. The first sound is short, flapping, weak, toneless, almost to extinction; the second weak and thin: there is no murmur.

The pulse, irregular in force, may be excessively so in rhythm,—no connection of time being traceable between it and the systoles of the heart.* Excessively feeble, fluttering, and small, it is not of necessity very frequent: there are instances, indeed, in which it has appeared unnaturally slow; this may have been deception, from the weak systoles not always impressing the distant vessels.

There are few symptoms that can be distinguished from those of dilatation. General languor, weariness, incapacity for exertion of any kind, failure of strength, sallowness and lividity of the face, feeble palpitation, protracted fits of dyspnœa, and painful sensations about the heart, occasionally actual angina, are common to the two affections. Dropsical effusions probably do not occur unless there be dilatation. But the fact is, softening of any duration is rarely unaccompanied with some increase, general or local, of capacity of the cavities. Softening is always a serious condition: it aggravates the ill effects of any other heart-affection it may coexist with, and has of itself proved suddenly fatal, with or without symptoms of angina.

III. The feebleness of impulse, without increased area of dulness, and the feeble irregular pulse, without the signs of valvular (especially of mitral) disease, are the points on which the diagnosis of softening turns; but these characters will not distinguish simple from fatty softening.

IV. In the *treatment* of the disease, an attempt must be made to improve the nutrition of the heart by raising the powers of the system generally, and improving the blood. Animal food, port wine in moderation, pure bracing air, regular-but very gentle exercise, and attention to the state of the skin, are the hygienic means indicated. To tonics, quinine, iron, the mineral acids, we must mainly trust among medicinal agents. Astringents (provided their ill influence on the bowels can be obviated) might be advisable—among the number, gallic acid.

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* In typhoid fever these characters of pulse, suddenly supervening, have been shown by M. Louis to be significant of *acute* softening of the heart.

Quietude, mental and bodily, is indispensable; too much exercise is infinitely worse than none at all; indeed, the patient's instinct, as well as his feebleness, lead him to disobey instructions that are sometimes given in such cases for forced pedestrian exercise.* Fits of passion should be studiously avoided. Direct sedatives of the heart cannot be given without extreme danger; and inhalation of æther or chloroform is under no circumstances permissible.

§ II. *Induration.*—General induration of the heart, excessively rare under any circumstances, scarcely occurs except from contractile hardening of solidified lymph and calcification. It may be supposed to weaken the muscular, and intensify and give undue clearness to the valvular, portion of the first sound. Should any knocking sound exist at the apex, it would probably be peculiarly clear. But these signs require revision at the bed-side: I give them hypothetically, having never met with a case of the kind.

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* It appears to me that error is very frequently committed in the attempt to force patients, labouring under various diseases, to *walk, walk, walk,* in spite, often, of their own convictions that each additional walk has helped them on towards that bourne, whence no *walker* returns. I do not refer, merely, to the bygone well-known practice in a central town of England,—but to the too indiscriminate recommendation of toilsome foot exercise by many of the profession generally. I have known a man labouring under chronic nephritis, with alkaline urine and the general cachectic condition of that disease, directed to perform such pedestrian feats, by way of recovering his health, as he himself well knew would, on the very first day, have brought on such an *acute* attack, as probably would have confined him to bed or a sofa for a month. I entertain not a doubt that the lives of sufferers from cardiac disease are often put in jeopardy by the effort to exercise beyond their powers. Because, in the *physiological* order of things, it is good to walk, the inference is at once drawn that it is universally good in the *pathological* order of things. But, in point of fact, each disease has its own appropriate kind and quantity of exercise,—and that kind and that quantity are (as all truths in therapeutics) to be learned in each instance by *experience* alone.

Induration of the papillary muscles in the left ventricle is not very uncommon: if combined with contraction of their substance, it must cause mitral regurgitation.

ADVENTITIOUS PRODUCTS.

§ I. *Calcification.*—Calcification may occur in any one of the tissues of the heart, in the actual substance either of induration-matter, fibrinous coagula, atheroma, or tubercle. In the valves it produces obstruction and regurgitation; in the cavities its effects fall in with those of polypoid concretions; in the pericardium, and still more in the heart's substance, calcification obstructs the heart's movements; in the coronary arteries, if carried to any extent, it must interfere with the nutrition of the organ, and may induce fatal angina.

Calcification of false membrane in the pericardium may conceivably give an osteal quality to the percussion-note over the heart, and impress something of a similar character on the systolic sound: here are conditions which, with a previous history of pericarditis, might give an inkling of the existence of the state in question. There are no known means of distinguishing calcification of the endocardium from other morbid changes, interfering organically with the play of the valves.

§ II. *Fat.*—I. Fat accumulates in three positions in connection with the heart—(*a*) under the pericardium, (*b*) under the endocardium, and (*c*) amid the muscular fibres.

(*a* and *c*) Sub-pericardial fat, chiefly gathering about the right side of the organ, and the ventricles rather than the auricles, when abundant, encroaches on, and to a greater or less depth renders the muscular texture beneath soft and atrophous. But this influence on the nutrition of the fibres is produced by simple pressure, not by intrinsic deterioration of the fibre itself.

Laennec was of opinion that this species of fatty affection "must exist in a very great degree before it gives rise to any *serious* complaint;" and he was probably right. True, cases have been recorded in some number, where the gravest effects have been assigned to such fatty disease; but very probably it must either in these instances have led to total destruction of fibre in limited places, or intra-sarcolemmous fatty change must have coexisted. There is no *proof* that rupture of a fatty heart has occurred, unless where the latter existed, though it very conceivably may have occurred independently of this.

Still fatty accumulation under the pericardium produces minor annoyances, and some physical signs. Sensation of oppression, or even pain, about the precordial region; syncopal feelings on exertion; inability to walk quickly on level ground and to get up hill, except with great and painful effort; inclination to coldness in the extremities; feeble (but, as far as I positively know, regular) pulse; sluggish action of the liver and bowels; occasional giddiness; and feeble cardiac impulse, with a too extensive dulness under percussion, the sounds, especially the first, being weak and toneless;—these were the symptoms and signs in the only person (a male, ætat. 64) I happen to have observed during life and opened after death, whose heart was at once loaded with sub-pericardial fat, and positively free from softening or intra-sarcolemmous oil. There was in this instance a considerable quantity of fat in the lower part of the mediastium, which may have contributed to weaken the heart's shock against the side. Hope speaks of the pulse being irregular in such cases; but in the only instance where he made a *post-mortem* examination, the tissue of the organ was softened as well as loaded with fat.

(*b*) Minute pellets of fat are sometimes seen under the endocardium: in the present state of knowledge they are devoid of clinical interest.

§ II. Fatty disorganisation of the proper muscular fibre is the most important change belonging to the present class. An organ thus affected loses consistence more or less, tearing in extreme cases with the greatest ease; varies in tint from a delicate faded-leaf hue to a pale dirty brown, mottled with darker spots; rarely feels greasy; may be natural in size, hypertrophous, or, as is much more common, dilated; infiltrated with blood, aneurismal or actually ruptured. The distinction of fatty degeneration and fatty infiltration, made by Laennec, has been confirmed by the microscope: oil globules may be seen within the sarcolemma; the oil is *intra-sarcolemmous*. Whether the appearance of oil is prior to wasting of the proper muscular element, or secondary to that change, is a point yet undetermined: Mr. Paget's observations rather raise than settle the question.

The physical signs are those of a soft heart. Weak impulse, indistinctness of the apex-beat, unchanged percussion-dulness (unless there be alteration of bulk from some other cause); a feeble, toneless, short first sound; a long first silence, and a feeble second sound (this may be of better tone at the second left than the second right cartilage, if the fatty disorganisation be, as it often is, in great excess in the left ventricle). Possibly a dynamic mitral regurgitant murmur may sometimes occur; but I do not know this from observation. The pulse is irregular in force and rhythm, either constantly or from time to time under excitement, the influence of flatulence, indigestion, effort, &c. On such occasions it may become exceedingly frequent: I have known it uncountable—in the main from frequency; in part, however, from irregularity. Infrequency of pulse, occasionally met with, is in some cases referrible to the weakness of occasional systoles; but the systoles are themselves sometimes much less frequent than natural.

Unable to undertake any sustained labour, exhausted on the first attempt almost; irritable in temper (this may come, in the poor, of their incapacity for work); easily put out of breath, and subject to fits of dyspnœa, but not constantly asthmatical; seized occasionally with palpitation, attended with choking sensations, cardiac uneasiness, pain, or actual angina; readily becoming faint on exertion, and falling into a state of actual syncope from time to time; suffering occasionally, too, from vertigo, dull aching sensations in the head, and somnolence; œdematous and livid in the lower extremities and face,—these patients are often possessed with the idea (and it occasionally proves a prophetic one) that they shall expire suddenly. Death has, in truth, occurred instantaneously, from rupture into the pericardium, by coma, and by syncope; in other instances, the fatal event is slowly brought about by asthenia. Fatty disorganisation of the heart is by no means necessarily fatal: I have known extensive destruction of the kind exist, where death had occurred from unconnected chronic disease of other organs; and the proofs that a slight superficial extent of the change may be comparatively innocuous are of daily occurrence. Nevertheless, could we positively diagnosticate the disease, no matter how slight its extent, its existence should never be lost sight of, either in regard of prognosis or therapeutics.

The treatment of the affection is precisely that recommended for softening of the heart.

§ III. *Tubercle.*—(*a*) Tubercle very rarely forms in the heart; and only in cases of general tuberculisation, or as an extension from similar substance deposited under the pericardium. Its signs and symptoms, if it have any peculiar to itself, are unknown.

(*b*) More commonly, tubercle forms beneath the serous layer of the pericardium (cardiac or parietal). Occurring in the state of semi-transparent grey granulation, it may remain latent, or excite inflammation of that membrane. The ensuing pericarditis produces the same physical signs and local disturbance as if it were of idiopathic origin. But, probably on account of the tendency to secondary formation of tubercle amid the simple inflammatory exudation-matter,* the disease, unless fatal at once, tends to lapse into a chronic state (just as peritonitis of the same origin), attended with more or less constant local symptoms. This was the view of Laennec; and it is supported by the course of a case that fell under my own notice.

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* Carswell's Drawings, U.C. Museum, A. 533. Fig. 2.

In ordinary phthisis, as appears from the records of M. Louis, tuberculous pericarditis is very 'rare: it is greatly more uncommon than pleuritis or peritonitis, and somewhat so even than meningitis, of that form.

The diagnosis of tubercle in the pericardium can only be made through the pericarditis it entails; true, it is infinitely probable, that miliary tubercles would themselves produce friction-sound, but in the present state of knowledge, the discovery would rather lead to a mistaken than correct diagnosis. And the tuberculous character of the pericarditis would not be announced by any peculiarity in its signs; still, an attempt at divining its character might be made in the following way. If there were neither rheumatism, nor Bright's disease, nor adjoining pleuro-pneumonia present, and the patient had received no injury to the chest, and was distinctly of strumous constitution, the probabilities would be much in favour of the tuberculous origin of the inflammation. And this, too, although there were no distinct pulmonary signs of advancing phthisis; for, herein resembling tuberculous peritonitis, this form of disease has mainly been observed in cases where the lungs were but slightly affected.

The determination of the *nature* of such pericarditis is not a mere matter of diagnostic curiosity. The most appropriate treatment, as Dr. Burrows has successfully argued (Med. Chir. Trans., vol. xxx.), can scarcely be that adapted to rheumatic pericarditis. Blood-letting should be had recourse to in extreme moderation; mercurials must be given with all needful care to avoid ptyalism. Blisters in the neighbourhood of the præcordial region, dry-cupping, moderate purgation, diuretics with iodide of potassium and alkalies, are the agents on which, in connection with antiphlogistic regimen, reliance must be placed. Animal food may be allowed earlier in this than in other varieties of the disease.

§ IV. *Cancer.*—The morbid anatomy and the general pathological laws of cancer of the heart and its membranes, especially of the former, are established with a fair amount of fulness and precision. But as I have, at the present day, only to repeat what I wrote some years ago,—the history of cancer of the heart, in respect of symptoms, diagnosis, and treatment, cannot with existing materials be established,—there is no motive in a practical work of this kind for dwelling on the subject.

§ V.—*Entozoa* infest the heart sometimes, both the acephalocyst and the cysticercus. An acephalocyst the size of a pigeon's egg is seen in the septum of the ventricles in the heart of a woman (U. C. Museum, No. 2293), who died suddenly while engaged in her household affairs. Among Dr. Carswell's drawings (U. C. Museum, A. 9), is the figure of a heart containing in the posterior part of the left ventricle a full-sized acephalocyst, which protrudes on the surface. The patient had died of phthisis, and the entozoon was accidentally found on the dissection of the body; and as she had died in hospital, and the heart had not attracted attention, there were, probably, no symptoms.

DISEASES OF THE ORIFICES.

I. Diseases of the orifices of the heart interfere in either, or both, of two ways with the circulation through the organ. They are constrictive in anatomical character, and hence produce *obstruction* of the onward current; or they entail disproportion between the size of the orifice to be closed and the valve to close it, and hence lead to *regurgitation* of a backward current. Each orifice is in theory capable of each kind of disease; but the frequency, with which the various orifices actually suffer, differs very widely. This question, however, requires to be considered in two points of view, the anatomical and the clinical: the excess of organic change in the actual texture of the valves and orifices of the left side, is very great; the excess of disturbance, clinically demonstrable, in the functional condition of the orifices comparatively much less. This comes of the frequency with which the tricuspid valve is incompetent, through mere widening, without textural change, of its orifice.

Obstruction of orifices, as far as is known, is always of organic character; and is caused by morbid change in the valves, in the wall of the orifice itself, or by extraneous pressure. Regurgitation through orifices is, certainly, in the vast majority of cases, likewise of organic character, and is caused by morbid change in the valve, in the wall of the orifice, or in some part of the apparatus connected with the closure of the valves. But it is highly probable that regurgitation may be produced dynamically in one orifice at least (the mitral), through functional imperfection of the closing apparatus, which is free from organic change. Regurgitation of this kind is of temporary duration, and does not produce serious results; so that, practically, the diseases of orifices may be regarded as organic and mechanical.

II. Diseased valves, diseased orifices, and diseased states of the apparatus effecting the closure of the valves, are, then, the causes of perverted circulation through the outlets and inlets of the heart. (a) *Diseased valves* are the causes of *obstruction* through local endocarditis and its products interstitial and superficial, alterations of form, and adhesions of the divisions of the valves *inter se;* through deposits of fibrine from the blood on their surfaces; through atheroma, calcification, and their sequences, and, in some instances, through hypertrophy. Diseased valves, again, are the source of *regurgitation* through endocarditis and its effects, (especially when these tend to produce puckering and diminished superficial size, or actual destruction of the divisions of the valves, or adhesion of these either to each other, or to the adjacent wall of the heart or great vessel to which they belong, or to produce thickening and shortening of the chordæ tendineæ, or rupture of these),—through fibrine deposited among, and interfering with, the action of the chordæ tendineæ,—through atheroma and its sequences,—through hypertrophy obstructing their fall,—and through atrophy in depth, rarely in substance by extensive perforation of the valves,—and through atrophy of the chordæ tendineæ. (b) *Diseased orifices* (the actual substance of the valves being sound) lead to *obstructions* through the products of endocarditis, atheroma, and calcification lying upon, or producing changes in, their surfaces or walls. And disease of the orifice is the cause of *regurgitation,* where the valve being sound, the opening is morbidly widened to such an extent as to render the former inadequate to its closure. (c) *Diseased states of the apparatus,* connected with the closure of the valves, are seen in induration and diminished bulk of the papillary muscles, and shortening of the cordæ tendineæ; they produce *regurgitation.* It may be made a question whether imperfect action of the papillary muscles, from disease of their substance, may not act obstructively also, and throw an obstacle in the way of the passage of the blood from the auricle to the ventricle.

III. Such are the general characters and immediate effects of the morbid states of the orifices and their connected apparatus. We will now pass to the consideration of the signs and symptoms of these morbid states at each orifice. These signs and symptoms, it must be noticed, are solely of mechanical origin; and hence their discovery discloses the existence simply of obstruction or regurgitation at a certain orifice, but tells nothing *directly* as to the nature of the morbid process (whether inflammatory, fatty, calcifying, &c.,) that has engendered the mechanical difficulty. In the majority of cases valvular disease entails some form of anatomical change in the substance of the heart itself; the proper signs of the former and of the latter are therefore met in frequent clinical association; but, for purposes of precision, it is necessary each class of signs should be enumerated apart,—as is done in the following description; the former under the head (a), the latter under the head (b).

Mitral regurgitation.—(a.) The impulse, in highly marked disease of this species, is irregular in force and rhythm; and systolic thrill may sometimes be felt at and about the left apex. The results of percussion are unaffected; and, to save repetition, it may here be stated once for all, that valvular disease *per se* never alters the area or intensity of the heart's dulnesses, superficial or deep-seated. The essential character of this regurgitation is systolic murmur of maximum force at the left apex, and possessing the other characters already enumerated, (*a*. p. 222). The systolic sound, completely or incompletely covered in this position by the murmur, may be perfectly natural at the ensiform cartilage and at the mid-sternal base; the second sound often weakened (in consequence of diminished calibre of the aorta) at the second right, or aortic, cartilage, is accentuated in many, but not in all, instances at the second left, or pulmonary, cartilage. Sometimes the second sound is distinct and sharp at the left apex, much sharper than at the pulmonary cartilage.*

Systolic murmur limited to the left apex is never of blood origin, as far as I have observed, therefore never chlorotic: the existence of chorea will distinguish the peculiar dynamic murmur of that disease: the murmur of this site and rhythm attending simple dilated hypertrophy will disappear under treatment. The diagnosis of murmur caused by mitral regurgitation and that produced by friction of blood against irregularities of surface at the base of the ventricle, cannot, I think, be made with positiveness in the present state of knowledge (*vide* p. 223)—but fortunately the latter kind of mechanism is excessively rare. I have already spoken of the influence of feebleness of the heart in preventing the evolution of murmur, of which the physical causes exist: when the heart is texturally feeble (as in fatty softening) its action may be excited by a few turns in a room, or by any other effort, and the murmur will then become audible; when it is feeble from collapse, this must be allowed to pass away, before a diagnosis is ventured on.

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* The case of Anne Gippin (ætat. 35, U. C. H., vol. v., p. 292: December 28th, 1850) gave a remarkable illustration of this fact. The mitral orifice was greatly constricted, the tricuspid dilated, and there was slight aortic regurgitation.

(b.) Dilated hypertrophy of the left ventricle is the common sequence of mitral regurgitation; hence the apex-beat and impulse generally are carried outwards and lowered somewhat. The impulse is increased in force, and in rare instances there is auricular impulse at the second interspace,—either pre-systolic, when it comes of hypertrophy of the auricle, or systolic, when it is communicated from the ventricle. The area of percussion-dulness is increased, especially to the left; but, from sequential hypertrophy of the right ventricle (which I have oftenest observed in early youth), extension of dulness may eventually take place to the right also.

The pulse presents itself in either of the following states:—Regular in force and rhythm; small, with occasional sharpness; rather frequent, and compressible, unless there be much hypertrophy; or irregular in force and rhythm, sometimes to an excessive degree, small, feeble, with occasional sharpness, and tremulous under excitement of the heart. In either of these states, the systoles and radial pulses may fail to correspond in rhythm and in number.

Pure mitral regurgitation produces but little effect on the systemic capillary circulation. The disease may exist for years without inducing either general dropsy or congestion. If dilated hypertrophy supervene, systemic obstruction occurs with a facility proportional to the amount of dilatation and the impoverishment of the blood. The symptoms then become those of dilated hypertrophy, aggravated, *plus* certain others, to be by-and-bye mentioned, more or less peculiar to the regurgitation. I use the word "aggravated" advisedly; it has not occurred to me to observe cases justifying the notion that mitral regurgitation may act as a sort of safety-valve to a dilated and hypertrophous left ventricle.

The disturbed conditions of the sensorial functions sometimes observed in mitral disease are likewise the results rather of the dilated hypertrophy, with which it is associated, than of itself alone; still, when carried to a great amount, it may secondarily, through its influence on the pulmonary circulation and the right heart, tend to congest the brain passively. No cases have fallen under my notice supporting the notion that colourless softening of the brain is a direct dependence on mitral regurgitation,—a notion very ingeniously advocated by Dr. Law.

The essential symptoms of mitral disease are pulmonary,—congestive and irritative. The current thrown back through the auricle on the pulmonary veins tends to congest the lungs; and it is not unreasonable to admit that the effort on the part of the right ventricle to overcome the increasing obstruction may induce irritation. Cough, watery expectoration, dyspnœa, and orthopnœa; actual bronchitis, pulmonary œdema, pneumonia (congestive or irritative), passive congestion and pulmonary apoplexy (commonly nodular), are the direct effects of the disease. The expectoration may by the latter condition be stained of dark blood tint, or blackish; or actual escape of pure blood may occur: what maximum quantity of blood may be brought up from this cause at a time, I do not know; I have never seen any large amount.

Mitral Constriction.—(a.) I have never observed diastolic thrill at the left apex, though it is conceivable that, if a highly hypertrophous left auricle lie behind the constricted orifice, the current may be rendered sufficiently strong to produce that thrill (p. 176). The impulse is irregular and unequal in force. The characters of the murmur of this disease will be found in a former place (*v.* p. 226): its frequent absence renders the positive diagnosis of mitral constriction far from easy. A natural second sound may be perfectly audible at the left apex, though there is marked mitral constriction, and even mitral constrictive murmur, present; but, so frequent is the association of regurgitation, that it is very rare to find a pure first sound at that point. At the mid-sternal base the first and second sounds are of natural character, except that they are sometimes feeble, from the smallness of the current sent on from the left ventricle. At the aortic cartilage, both sounds are feeble; at the pulmonary, both, on the contrary, and especially the second, are full and accentuated: if there be co-existent insufficiency of the tricuspid valve, this accentuation may, however, be prevented from occurring.

(b.) Left auricular pre-systolic impulse, and right ventricular impulse, are frequently found as co-existences, in consequence of dilated hypertrophy being gradually produced in those situations. The pathological reasons of these enlargements are sufficiently clear. The area of percussion-dulness, especially to the right side, will on similar grounds be increased.

The character of the pulse and the pulmonary symptoms are the same as in regurgitant disease of this orifice. When constriction and regurgitation co-exist, as is commonly the case where there is constriction, either the murmur of regurgitation is heard alone, or a double to-and-fro murmur is distinguished; its two portions differing in quality and pitch.

Tricuspid Regurgitation.—(a.) I have never known tricuspid regurgitation productive of thrill; the characters of its attendant murmur (which, for reasons there assigned, is often absent) are set down in a previous page (β. p. 224). The second sound is weak at the right apex, almost to extinction sometimes. Both sounds at the base and at the aortic cartilage may retain their natural characters; if the disease be pure, the second sound is weak at the pulmonary cartilage.

(b.) Commonly coincident with dilatation, simple or hypertrophous, of the right ventricle, the signs of these conditions are discernible:—Epigastric and right sternal impulse, of variable force, but out of proportion with that on the left side (unless the left ventricle be accidentally hypertrophous also), and increased area of percussion-dulness, mainly to the right. Hypertrophy of the right ventricle will prevent the enfeebling of the second sound appertaining to the pure regurgitation.

The arteries, which tricuspid insufficiency may be supposed to affect directly, (the pulmonary) cannot be felt; and there is nothing peculiar in the pulse of those derived from the aorta. The cervical veins distended, knotty, pulsatile, and refilling readily from below, may be the seat of thrill. But all these various signs are only found prominently in well-marked cases; if the regurgitation be slight, or if the right ventricle be very weak, pulsation, and thrill especially, may be altogether absent.

The pulmonary capillaries escape congestion in this form of disease. That the systemic capillaries, on the other hand, are essential sufferers, has been ably demonstrated by Dr. Blackiston: however, as I have already stated (p. 478) the connection is not an absolutely necessary one; and dilatation of the cavities must, independently of regurgitation through any orifice, be admitted to play a most important part in inducing systemic stagnation. For the effects of this systemic stagnation, I would refer to the section on Dilatation.

Dr. Blackiston's results tend powerfully to show that obstruction of the cerebral capillary vessels and apoplexy are much more frequent in cases of heart-disease with, than without, stagnation of the *systemic* capillary circulation: hence the inference, that cerebral obstruction and apoplexy are more connected with the heart through dilatation and tricuspid regurgitation than through hypertrophy of the left ventricle.

Tricuspid Constriction.—Constriction of the tricuspid orifice would theoretically give a diastolic murmur of maximum force at the ensiform cartilage. But this condition of the orifice is excessively rare, because atheroma and calcification are themselves extremely uncommon in this situation; and an amount of coarctation reducing the orifice to the size of the middle finger may not, as a case observed by Hope proves, give rise to murmur,—probably on account of the weakness of the current.

Aortic Constriction.—(a.) In well-marked cases, systolic basic thrill may be caught; and the murmur already described (*v.* p. 225) exists. The natural first sound of the heart is audible at the left apex, though slightly covered by the basic murmur: the second sound, weak at that apex, at the base, and at the aortic cartilage, may be slightly murmurish at the two latter points, in consequence of some regurgitant tendency. The second sound is occasionally reduplicate at the base.

The murmur of chronic aortic constriction is one of those open to the greatest number of imitations. Of acute endocarditis, pressure by pericardial fluid or false membrane, as causes of similar murmur, I have already spoken; and, further, the distinction of this murmur from that produced by disease of the aorta close to the valves, and by abnormal communications within the heart, is far from easy. It has already been shown that displacement or twisting of the heart on its axis does not necessarily produce murmur, though this sometimes occurs. Of the blood changes, producing systolic basic murmur, spanæmia is the most important; its distinctive characters have already been inquired into (p. 219).

Weakness of action of the heart, or extreme smoothness of the constricted orifice, may prevent the development of murmur.

(b.) The impulse and altered percussion-sound of hypertrophy, with or without dilatation, of the left ventricle, are the common coincident signs. It sometimes happens that the murmur of mitral insufficiency is produced by the dilatation of that orifice following on dilatation of the ventricle.

The pulse, in cases of moderate coarctation, is not materially affected; if the constriction be great, the pulse, though regular in force and rhythm, is small, hard, rigid, concentrated; hardness and force signify hypertrophy behind the narrowed orifice.

Aortic constriction exercises no direct effect on the pulmonary capillaries; some indirect tendency to stagnation arises from the difficulty experienced by the blood, flowing from the pulmonary veins, in its entrance into a ventricle, which, again, has itself great difficulty in discharging its contents.

It is truly remarkable to what an extent this coarctation may be carried, without producing systemic stagnation: the opening may be no larger than a pea, without leading to the very slightest œdema, even of the ankles. This immunity, however, only holds as long as the capacity of the ventricles and the width of the tricuspid orifice remain unaffected; if the blood becomes spanæmic, too, anasarca occurs independently of these latter changes. It has been conjectured that the peculiarity depends on the slowness of the circulation in old people; but the circulation is not always slow in old people, and young adults, with constriction of the aortic orifice, also remain free from systemic dropsy.

It seems theoretically probable that a constricted aortic orifice will weaken the effect of a hypertrophous ventricle on the brain.

Aortic insufficiency.—(a.) Diastolic basic thrill, though it has not fallen under my notice, may conceivably be perceptible, especially if the blood be at all spanæmic. I refer to a previous page (*v*. p. 227) for the characters of the murmur of aortic insufficiency. The state of the heart's sounds is as follows:—The first, at the base, may be natural or very nearly so, dull, obscured by a soft murmur, or masked totally by a harsh one. The second sound, at the left apex, may be null, faint, or distinct and sharp; in the latter case, the sound heard is either that of the pulmonary valves transmitted, or is produced by fall of the aortic blood, during regurgitation, into the ventricle below (*vide* p. 197). At the aortic cartilage the first sound is of variable character; the second, a murmur, more or less marked. Both sounds may be perfectly natural at the pulmonary cartilage, and occasionally the second accentuated, if there be no mitral regurgitation.

Aortic regurgitant murmur is, as far as I know, constant, where its physical cause exists; weakness of ventricular action obviously cannot have the same effect in rendering a regurgitant, as a direct, murmur obscure. If a double aortic murmur exist, the systolic portion is best transmitted upwards in the course of the vessel, the diastolic generally downwards in the line of the sternum,—the latter peculiarity probably owing to the direction of the current and the aspirated quality of the murmur. The infrequency of direct tricuspid murmur and the coexistence of other signs of aortic regurgitation, will prevent any error arising from the prominent character of the aortic murmur at the ensiform cartilage. It is conceivable that pressure on the outer orifice of the aorta just above the valves, might interfere with the closure of these, and so produce regurgitant murmur; in a case of displacement of the heart by pleuritic effusion, diastolic murmur existed under circumstances tending to connect the murmur with the displacement (*vide* p. 286). I have never met with a positive example of aortic *blood-murmur* diastolic in time, but such a murmur may be simulated in the following way:—spanæmia exists, with a strong systolic basic murmur; at the same time, deep-seated hum is present in the pulmonary veins; this hum is covered during the systole by the strong, blowing aortic murmur, but becomes audible during the diastole, when there is no aortic murmur to interfere with it.

(b.) The concomitant signs are those of hypertrophy, eccentric or simple, of the left ventricle; frequently of aortic constriction, and occasionally of mitral regurgitation.

The pulse is sudden, abrupt, short, jerking—a sudden fall back following instantly the rise of the vessel. Commonly regular in rhythm, sometimes notably lagging behind the systolic sound of the heart (p. 206), occasionally bisferiens, each pulsation is, in rare instances, attended with thrill. Of visibleness and locomotion of the superficial pulses, enough has already been said (p. 238).

Aortic regurgitation affects the pulmonary circulation, either indirectly through mitral regurgitation (of which it is itself the original cause), or more directly through the embarrassment produced by the collision of blood, falling from the aorta, with blood coming forward from the auricle. The tendency to sequential hypertrophy of the left ventricle (mainly, I think, about the apex in these cases), does not, as in aortic constriction, afford any help to the circulation; on the contrary, by increasing the amount of distension of the aorta at each ventricular systole, it intensifies the force of the succeeding recoil.

The cerebral capillaries can only be affected secondarily through the pulmonary class; and the systemic capillaries are much in the same position. Regurgitation may exist to the highest amount without a particle of œdema of the extremities.*

Pulmonary Constriction.—Constriction of the pulmonary orifice exists with some frequency in cases of cyanosis, but coupled with such other conditions as render the attempt to fix on its special signs and effects difficult almost to impossibility. On the other hand, isolated constriction of the pulmonary orifice is necessarily rare, in consequence of the rarity of atheromatous and other changes in its valves. The characters of the attendant murmur will be found elsewhere (δ. p. 225). Hope teaches that a pulmonary constrictive murmur seems closer to the ear than an aortic, and on a "higher key, ranging from the sound of a whispered *r* towards that of an *s*;" the reason being that the pulmonary artery is nearer to the surface of the chest than the aorta. As regards closeness to the ear, his statement is, doubtless, correct; as regards higher pitch, the reason assigned for this is obviously erroneous; and, indeed, Hope himself unconsciously admits this, by adding that he has known the murmur "fall below *r*, when the circulation was feeble and slow and the obstruction slight."†

It is difficult to conceive that pulmonary constriction can exist to any extent without inducing dilatation, with more or less hypertrophy of the right ventricle, and systemic venous obstruction of a mechanical character.

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* *E. g.* Case of E. Barnes, U. C. H., Males, vol. v., p. 216.

† Of three cases, given by Hope, in illustration of the murmurs of pulmonary constriction and regurgitation, one only terminated fatally, and here (Op. cit. p. 598) the notes only state "there was a murmur over the semilunar valves." Hence his volume gives no positive information on the subject. That a systolic murmur in an anæmic woman, is louder at the pulmonary than the aortic cartilage, does not by any means prove, as he appears to think it does, that the orifice of the aorta is not the seat of the murmur.

Pulmonary insufficiency.—The theoretical characters of the murmur, significant of this state, have been already given (*θ.* p. 228). No visibleness of the superficial pulses of the limbs would accompany a basic diastolic murmur of this origin. By a singular fatality, while a certain number of examples of such destructive disease or insufficiency of the valves, as must have led to full regurgitation, have been observed *post mortem* in this country, in not one, that I know of, had the physical signs been clinically established. Theoretically, the effects on the systemic and cerebral capillary circulation must be most serious; and a sensation of dyspnœa, arising from the smallness of the quantity of blood actually reaching the lungs by each systole, might (unless the force of habit would counteract this influence) be expected.

IV. In fixing on the seat of production of any given murmur, the first point for the observer to establish is the position of the heart itself: if this be abnormal, as it very often is in consequence of changes in its substance, allowance must be made for its influence in altering the maximum position of murmurs. Now, the general tendency is to some form of enlargement of the organ, and as enlargements lower it, the maxima points of murmurs are very commonly slightly lower than the maxima points of the corresponding sounds in health. Certain conditions of the aorta or pulmonary artery, of the lungs, pleura, or mediastinum, also throw murmurs into unnatural sites, by changing the position of the heart. Pleural fluid accumulations have more influence on apex than on basic murmurs, for an obvious reason.

The state of the heart's substance also may exercise a direct influence on the intensity of murmurs. Thus, hypertrophous texture, lying in the course of the circulation, behind a murmur, will intensify this, if it be direct; exercise little effect on it, if it be regurgitant: take the instance of an hypertrophous left ventricle with severally a constrictive and a regurgitant aortic murmur. Hypertrophous texture, lying in front of a murmur, is without effect upon this, if it be direct; intensifies it, if it be regurgitant: take the instance of an hypertrophous left ventricle with severally a constrictive and a regurgitant mitral murmur. Dilatation and weakening affections of the heart's substance weaken any direct murmurs through orifices on which such substance may play. Dilatation lessens the force of a regurgitant murmur, in so far as power of backward propulsion is concerned; but as, on the other hand, it allows of considerable accumulation of blood for regurgitation, the weakening influence is somewhat counterbalanced.

The physical conditions of a given murmur positively existing at a given orifice, the character of that murmur may be seriously affected by the anatomical state of other orifices. Thus, aortic constriction may be almost murmurless, if there be any great co-existent mitral regurgitation, the aortic current is so seriously weakened by such regurgitation.

Of two murmurs produced synchronously at different orifices, one may mask or cover the other. An aortic constrictive and a mitral regurgitant murmur may exercise this influence on each other; and a mitral regurgitant may completely drown a tricuspid regurgitant. In the interspace between the maxima points of two synchronous murmurs, however, a spot may generally be caught where the intensity of murmur is less than at either, and the quality different.

An hypertrophous and widened state of the aorta will intensify its own regurgitant murmur; and if slight pouching exist at the sinuses of Valsalva, the rippling direction thereby given to the current will intensify both a direct and a regurgitant murmur. The state of the blood, too, is important: spanæmia greatly strengthens the force of murmurs of all kinds. The pulmonary veins and superior cava may be the seat of spanæmic murmur, simulating especially systolic apex and diastolic basic, cardiac murmurs.

Again, modified conducting power of adjoining textures may lead to error: thus an aortic regurgitant murmur may be heard better to the left than the right of the sternum, if the left lung be solid and the right emphysematous.

V. The more important *symptoms* of each variety of valvular disease are to be directly gathered from the statements made as to the influence of each on the different capillary systems. There are a few which may be considered common (though still not in precisely similar amounts) to all of the class. Wasting of strength, comparatively little of flesh, and sudden starts from sleep, which is habitually disturbed by frightful dreams, are very generally observed. Pain in the cardiac region is sometimes present, varying in amount from a slight aching sensation to the intensity of angina. It has been suggested that atheromatous and calcified conditions of the valves and orifices, interfering with the facility of stretching of these parts, may explain this. Palpitation is a symptom (except in rare instances) common to all valvular diseases carried to any amount. An albuminous state of the urine I have found most connected with tricuspid regurgitation; as also engorgement and œdema of the female pudenda and passive lucorrhœa.

VI. The mean duration of the various valvular diseases is quite unknown: even the most serious are not incompatible with prolonged life, though sudden death (by syncope or coma) may occur during the course of any one of the number. In respect of comparative severity and danger, the chief valvular derangements may be placed in the following descending series: —tricuspid regurgitation; mitral regurgitation and constriction;* aortic regurgitation; and, least serious of all, aortic constriction.

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* Hence the danger of valvular diseases, as a class, is not to be estimated by the amount of murmur they habitually entail,—for tricuspid regurgitation and mitral constriction are precisely the two with least constant murmur. This is sufficient proof that implicit faith—nay, any faith—in respect of prognosis, must not be put in the conditions of valvular murmurs; and it may be well here, again, to remind the younger reader of what may be fully gathered from the description in the First Part, that there is no direct connection between the amount of disease at an orifice and the intensity of an existing murmur;—the very *weakness of a murmur may, indeed, be a fatal sign.*

Dilatation of the heart always renders any valvular condition more dangerous; and, as a rule, hypertrophy, though to a less degree, has the same kind of effect. Hypertrophy of the left ventricle, however, and directly as its purity, may mitigate the effects of aortic constriction. I do not know from experience the influence of such hypertrophy on regurgitant disease of the aorta, and theory speaks in both ways.

VII. Whatever be the mechanical condition of diseased valves, and whatever be the nature of the diseased action (inflammatory, fatty, calcifying,) that has impaired their freedom of function, they lie themselves without the pale of direct *treatment.* Valvular disease once chronically established cannot be cured: we can neither remove deep-seated induration-matter, atheroma, nor calcareous substance, nor lengthen tendinous cords that have been shortened by morbid processes. Hence the importance of at once guarding as much as possible against one of its frequent causes, acute articular rheumatism, and of preparing for the active treatment of acute endocarditis the moment there is reason to suspect its immediate advent.

But though these diseases be anatomically incurable, their worst functional effects may be long, in some cases indefinitely, averted, by measures accordant with a common-sense view of their nature and ratified by experience. Whatever be the valve implicated, the treatment is directed not towards its own disease, but towards the moderation or prevention of hypertrophy of the muscular substance of the heart or dilatation of the cavities, and the removal or relief of any symptoms that may arise. Practically the treatment of valvular diseases comes to be that of hypertrophy or dilatation, due regard being had to the influence exercised by the mechanical obstruction on the character and tendencies of those conditions of the heart's substance. Thus, if with valvular disease there co-exist dilated hypertrophy, occasional moderate venesection, or the abstraction of blood by cupping or leeching over the pericardial region, will be advisable: more so in cases of superadded mitral regurgitation than of aortic constriction, for reasons easily inferrible from the influence of such hypertrophy in these two valvular diseases. Profuse blood-letting is, under all circumstances, absolutely improper: it cannot remove either the disease of the valves or of the heart's substance, and may induce anæmia, excitement of the heart, and early dropsy. If dilatation exist, with or without attenuation, venesection is decidedly contraindicated: three or four leeches may, however, even under these circumstances, be applied to the cardiac region for the relief of palpitation, anginal feelings, or sudden engorgement of the right cavities. Dry-cupping and mustard poultices should, however, first be tried: they often give quite as much relief, and without any concomitant sacrifice.

Of the value of *diuretics*, when dropsy has appeared, no doubt can be entertained; but long before matters have proceeded to this point, medicines of the class seem useful, by diminishing the quantity, without impoverishing the quality, of the circulating fluid, and so lessening the tendency to clogging of the intra-cardiac circulation and the proneness to palpitation. The acetate, nitrate, iodide, and bitartrate of potass (the latter in two drachm doses largely diluted), nitric æther, compound tincture of iodine, the infusion and spirits of juniper, the decoction of chimaphila, and other agents of the class may be variously combined and successively employed. The action of these diuretics is facilitated by occasional small doses, at bed-time, of blue pill and squill. When the kidneys are much congested, removal of that congestion must be effected, wholly or partially, before the organs will act. Cupping to a small extent or dry-cupping in the renal regions, sometimes exercises a remarkable effect in facilitating the action of diuretics by removing congestion: in the same way is to be explained the favourable influence of mustard poultices, and even *blisters* to the loins. Urine, more or less impregnated with albumen, previous to these measures, becomes perfectly free (to ordinary tests) of that principle after their employment.

Regularity of alvine discharge, in all diseases important, is very essential in these cases, to prevent engorgement of the liver, and obviate the necessity for effort in the act of defecation. Purgation is to be avoided prior to the occurrence of dropsy. Dropsy once established, hydragogue purgatives (combined with diuretics) become the most important agents. Elaterium,* gamboge, bitartrate of potass, and the pulvis jalapæ compositus are the most valuable of the class. Stimulants may be requisite during the action of these medicines; and their action may be so rapid, that the mere loss of fluid shall weaken vitally and mechanically. There is no period of valvular disease at which the removal of dropsy may not be accomplished; but when the powers of the patient are seriously enfeebled, great caution, as regards the rapidity of that removal, is called for.

Saline *diaphoretics* aid the action of diuretics: the vapour-bath sometimes very perceptibly lessens anasarca.

If at any time the stomach be loaded, and its replete state excite palpitation, an *emetic* seems clearly indicated: the sulphate of zinc is the fittest. But where the circulation is very much embarrassed, emetics sometimes increase considerably that embarrassment, and unhappily it is far from easy to fix beforehand the probable influence of an emetic in any particular case. If there be doubt, it is better to refrain and allow the stomach to free itself gradually in the natural manner. Hope was of opinion (and with much *à priori* reason in his favour) that a state of prolonged nausea, by causing languor of the circulation, promoted the formation of fibrinous coagula within the heart.

Where the lungs tend to engorgement, especially in mitral disease, *expectorants*, either ipecacuanha, lobelia, stramonium, or squill and ammoniacum or senega, according to the active or passive character of the symptoms, become necessary. Dry-cupping of the chest, sinapisms, and blisters materially relieve such engorgement. *Anti-spasmodics* are necessary in paroxysms of dyspnœa, chloric æther, ammonia, &c. *Opiates* cannot be dispensed with at night in advanced cases; but where the valvular obstruction is considerable and the heart weak, caution is required in their exhibition. *Anti-acids*, carminatives, and light bitters, and other stomachic medicines, relieve the gastric discomfort so common in the victims of valvular disease; but the latter must not be permitted to an extent to stimulate the appetite to any great amount.

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* The formula I employ is as follows:—℞ Extract. Elaterii, gr. $\frac{1}{6}$-gr. $\frac{1}{2}$; Creasotonis, gr. i.; Extract. Hyoscy., gr. ii.; pro pill., i.

The propriety of administering mineral tonics will, in the main, turn on the condition of the heart's substance,—whether dilated or hypertrophous,—a subject already considered. Theoretically, invigorating tonics are more advisable in aortic constriction than in mitral regurgitation. Anæmia peremptorily calls for iron.

Issues or *setons* to the præcordial region sometimes relieve local pain and discomfort; they are otherwise valueless. At the very earliest period of chronic inflammatory changes in the valves, it is possible that ioduretted frictions may promote absorptive action.

In regard of diet, no constantly applicable rules can be laid down, except that moderation is important both in solids and, especially, in fluids. Exercise should never be pushed to fatigue, and laborious efforts of all kinds systematically avoided.

I have not spoken of digitalis. The action of this medicine, when it really does slacken the circulation materially, is rarely demonstrably beneficial, sometimes seriously mischievous, always hazardous. The obstruction to the circulation through the heart, necessarily produced by valvular disease, tends to promote coagulation of the blood within it; and on the evils of that coagulation it is needless to insist. If digitalis be employed at all, it is least dangerous, in its diuretic form, that of infusion.

It may appear strange that in this brief history of valvular disease, now brought to an end, so much importance should be attached to tricuspid regurgitation,—a phenomenon which, we are assured by some experimentalists, is constant in the state of health. Hope conceived that the absence of tricuspid regurgitant murmur in healthy persons sufficiently disproved the possibility of any such healthy regurgitation: his argument was, however, valueless, for, as is well known, highly-marked morbid regurgitation even may exist without any murmur. But I agree with Hope in his refusal to admit the reality of regurgitation in health; my grounds are, that if regurgitation existed, it would visibly affect the venous circulation in the neck, and that the experiments on which the doctrine is founded do not represent what common sense leads us to suppose must be the state of action at the tricuspid orifice during life.

MALPOSITIONS.

I. Malpositions of the heart are *congenital* or *acquired.* Of the *congenital* class the extra-thoracic, cephalic, and abdominal ectopiæ are totally without clinical interest. Not so, congenital malposition of the heart in the right half of the thorax; for this being a kind of displacement producible at all periods of extra-uterine life by a variety of diseases, it becomes of importance to have a means of positively distinguishing the malposition of congenital origin. Now this means is furnished by the position of the liver and spleen; for, except in very very rare instances, when the heart has formed and grown on the wrong side of the spine, the abdominal viscera, also, have been transposed. Where this guide is wanting, the distinction may be very difficult; and the observer is only justified in pronouncing the mal-position of the heart to be congenital after he has succeeded in excluding every possible morbid source of acquired displacement.

II. The diagnosis of acquired displacements is to be made by the position of the apex-beat and the impulse of the organ generally,—also by the comparative intensity of the sounds in different parts of the chest. The practical interest of these displacements (they are examined in a former place, p. 151.) depends upon the light thrown by their discovery on the diagnosis of various intra-thoracic diseases. They themselves produce no symptoms; and the restoration of the organ to its natural position can of course only be effected by removal of the condition displacing it.

CYANOSIS.

I. Cyanosis, morbus cæruleus or blue disease, are the names applied to a symptomatic state, prominently characterised by blue discoloration of the tegumentary membranes, and known by experience to be connected with various malformations of the heart and perverted modes of origin of its great vessels. The general tendency of the more common of these malformations is to alter the relationships naturally subsisting between the two sides of the heart and the two kinds of blood—dark and florid. But some of the number act in other ways, and the entire series may be referred, as species, to three classes, in the following manner:—

A. *Conditions causing direct communication of the arterial and venous circulation.* (*a*) *In the heart.*—Open foramen ovale; deficiency of part of the ventricular septum; perforation, of ulcerative or other character, throwing the auricles or the ventricles or all the four cavities into, practically speaking, a single cavity; heart formed of one auricle and one ventricle, the latter giving off one artery, which divides into a pulmonary artery and aorta, &c. (*b*) *In the great vessels.*—Freely pervious ductus arteriosus; aorta rising from both ventricles, or from the right ventricle, or from a trunk common to itself and the pulmonary artery, &c.

B. *Conditions causing distribution of black blood almost solely to the systemic capillaries, and of red blood to the pulmonary capillaries, without, practically speaking, any intermixture of the two kinds of blood.* Here appear cases where the aorta rises from the right, and the pulmonary artery from the left, ventricle, —the venæ cavæ, as in the natural state, communicating with the right, and the pulmonary veins with the left, auricle;—where, consequently, there are two distinct circulations, communicating alone by the ductus arteriosus, if this remain open.

C. *Conditions obstructing the entry of blood into the lungs, or intensely congesting them, so as to prevent oxygenation.* (*a*) *In the heart*:—Excessive smallness of the right ventricle; extreme narrowness of the tricuspid orifice: these states disturb the process of distribution of blood *to* the lungs. Great contraction of the cavity of the left ventricle, great coarctation of the mitral orifice: these conditions prevent the return of blood *from* the lungs. (*b*) *In the great vessels*:—Partial or complete obstruction of the orifice of the pulmonary artery.

II. The *symptoms* of Cyanosis are blue, leaden, purplish, violet, or almost black discoloration of the skin; the discoloration may pervade the entire surface of the body, but it is always most marked at the internal canthi, lips, nose, ears, below the eyes, and at the tips of the fingers and toes. Changeable in intensity from time to time, it is greatly deepened by palpitation, emotion, dyspnœa, and all conditions throwing any additional obstacle in the way of the circulation; generally speaking, the tint lightens materially after death. The ends of the fingers, if life continue for any length of time, grow more or less bulbous; in adults this peculiarity of form is sometimes singularly well-marked; at the same time the nails are incurvated; and the patients are said to be particularly subject to whitlow. The temperature of the surface and mucous canals, at their orifices, is low; the patients are constantly chilly, and sensitive to the least fall in the thermometer. Œdema of the feet sometimes exists; the frame is weak, the muscular and adipose systems ill-nourished. Nevertheless, the viscera may be of full size; their colour does not seriously differ from that of health,—but this may in great measure depend on their tint, as well as that of the skin, changing after death.

Though rarely the subjects of serious habitual dyspnœa, cyanotic persons frequently suffer from paroxysmal difficulty of breathing—often the result of disturbed circulation caused by fits of petulance and passion, to which they are, above the average, prone. Cough, either dry or attended with watery expectoration, probably through œdema of the lungs, frequent attacks of congestive bronchitis, added to, sometimes, the physical signs of emphysema, constitute the sum of pulmonary symptoms and morbid states. Palpitation active, tumultuous, with strong diffused impulse, a pulse ranging from 120 to 160, irregular in force and rhythm, often coexists with the dyspnœal paroxysms above referred to; syncopal tendency and actual syncope, though rare, do sometimes actually occur; the eyes are prominent, the expression wild, and the arms are tossed violently about. There is no particular pulsation observable in, even, the most deeply-coloured parts. Convulsions, somnolence, and semi-coma occasionally occur; and reflex phenomena, such as jerking action of the limbs and grinding of the teeth, are frequent during sleep.

The physical signs in the cardiac region vary according to the anatomical condition. In the most intense form of cyanosis, where the whole system is fed with very little else than black blood (as in transposition of the aorta and pulmonary artery), there need not of necessity be any abnormal physical sign at all. In the most common anatomical state (open foramen ovale and constriction of the orifice of the pulmonary artery, with sequential hypertrophy, simple or eccentric, of the right ventricle), systolic basic thrill, doubtless in the pulmonary artery, may be felt; the systolic murmur of pulmonary constriction may be traced up to the second left cartilage; and the signs of an hypertrophous state of the right ventricle will be coincidently found. I do not know that we are in a position to assert positively, that patency of the foramen ovale will in itself cause murmur; for in all records that I have met of the coexistence of the two things, either constriction of the pulmonary orifice is actually stated to have existed, or it may have existed—all mention of the vessel being omitted by the narrator. I once met with a systolic murmur in a cyanotic child, audible at a little distance from the surface; I saw the boy but once, and unfortunately neglected to note the topography (if I may use the expression) of the murmur.

II. Cyanosis is not always congenital. Of seventy-one cases, forty only existed at birth.* When appearing for the first time in early childhood, possibly enlargement of the heart gradually opens out the foramen ovale, and so induces the intermixture of the two kinds of blood; or perhaps congenital constriction of the pulmonary artery increases. In the adult, the appearance of cyanosis has sometimes been traced to a blow, a fall, or an effort; possibly some forcible separation of the edges of the foramen ovale had occurred; sometimes ulcerative openings in the auricular or ventricular septum have proved the immediate cause.

III. The only affection in the new-born infant with which it appears possible to confound cyanosis, is *apoplexia neonatorum* (intense congestion, with extravasation of blood, of the membranes of the brain and spinal cord), characterised by great lividity of face, swollen scalp, feeble action of the heart, slow, irregular respiration, clenching of the hands, convulsive actions, torpor, chilliness—conditions lapsing into fatal asphyxia, unless treatment (especially bleeding from the umbilical cord) prove successful. But the tint of skin in cyanosis is different, blueish, not livid; the scalp is not swollen; nor is there general tumidness of the upper part of the body; the action of the heart is rather in excess than deficient in strength; and the respiration is not laboured, irregular, and slow.

IV. It would be altogether beside the purposes of a work like the present to discuss the theory of cyanosis; but in justification of the influence ascribed, in the classification above given, to intermixture of venous with arterial blood, it must be stated, that the doctrine of venous stagnation adopted by Morgagni and

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* Stille; Amer. Journ. Med. Sc., N. S., viii.

M. Louis, to the exclusion of ordinary ideas concerning that intermixture, seems scarcely satisfactory. How is that doctrine reconcileable with the fact, that the most intense venous obstruction may arise in the adult without inducing true cyanotic discoloration? How comes it, too, if communication between the two sides of the heart be so unimportant, that, in five only of seventy-one cases of cyanosis collected by Stille (loc. cit.) was such communication wanting? Is it not likely that two things, so constantly found together, act as cause and effect; and that, where a widely open foramen ovale has been found (as it certainly occasionally has) without previous cyanosis, some corrective condition, either organic or dynamic,* has existed, to prevent the intermixture? Doubtless constriction of the orifice of the pulmonary artery will increase the darkness of tint, by inducing venous stagnation; but I do not think there is evidence to show that, unassisted, such constriction can produce cyanosis.

V. The treatment of a case of cyanosis resolves itself into the prevention, as far as possible, of paroxysms of dyspnœa and palpitation. Tranquillity of the circulation, by the avoidance of all emotional excitement, mental or bodily, and of all conditions likely to congest the lungs, the liver, and the abdominal organs, is to be aimed at; the temperature of the skin maintained by warm clothing, moderate exercise, and friction; and that of the body, generally, raised, if the stomach be not disordered thereby, by the free consumption of oil, fat, gum, and other aliments of respiration.

RUPTURE OF THE HEART.

§ I.—I. Rupture of the heart's substance into the pericardium, from all causes, spontaneous and other, indiscriminately, takes place with much greater frequency in the left ventricle than any other part of the organ; * but, if cases originating in external violence alone be considered, the right ventricle has, according to existing records, suffered more frequently than the left, in the proportion, according to Ollivier, of eight to three.

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* If the pressure of the current on either side of the opening be equal, there is no reason why each current should not pass on without commingling at all, or more than very slightly, with the other. Such nicely-balanced pressure must be rare, however, as the right auricle tends to become dilated and somewhat hypertrophous; and so, precisely, it is very rare that the foramen ovale is open to any extent without intermixture of the two bloods, and consequent cyanosis. It is probable also that a pretty free admixture is required.

Variable in size and form, sometimes smooth, sometimes ragged at the edges, the inner and outer openings—that is, the pericardial and the endocardial—may correspond or not; in the latter case, a sort of sinus exists in the wall of the ventricle, connecting the two. There is usually a single rupture only, but so many as five have been seen; sometimes a single opening on the inner surface of the ventricle communicates with two or more on its pericardial surface. The strata of muscular tissue next the endocardium, again, may be pretty extensively destroyed, while a tiny perforation only in the pericardium can be discovered. Although there is every reason to believe that, in the great majority of cases, rupture of the heart is perfectly sudden in its occurrence, it is clearly a gradual process in some rare cases; for instance, where hæmorrhagic softening of tissue has led to its occurrence: under all circumstances, the final breakage of the last few muscular fibres and outer serous membrane, that separate the pericardial and endocardial cavities, is instantaneous. The fissure runs very nearly three times as often parallel to, as at right angles with, the main fasciculi of the heart's fibres.

II. Considerably more frequent in males than females, in the ratio of 36 to 16, rupture of the heart is favoured by advanced age: it becomes comparatively frequent after the fiftieth, still more so after the sixtieth year. Immediately induced by efforts of some kind, by fits of passion, by great and abrupt thoracic congestion (as through sudden immersion in the cold bath), by blows and other injuries to the præcordial region, the way is

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* In 52 instances of rupture collected by Gluge (Path. Anat.) the left ventricle was the seat of the rupture in 37 cases; the right ventricle in 8; both ventricles in 2: the right auricle in 2; the left auricle in 3.

paved for its occurrence by various textural changes of the heart's substance or aorta: as fatty accumulation under the pericardium, intra-sarcolemmous fatty change; softening of undetermined kinds; dilatation with attenuation; local suppuration, ulceration, possibly gangrene; hypertrophy, with, probably, fatty change superadded; hydatids; calcification of the ruptured tissue; aneurism of the left ventricle; local destruction of the endocardium; apoplexy of the heart, (the question has, however, been raised, whether in cases where blood infiltration and rupture have been found together, the former may not have depended on gradual advance of the latter); coarctation of the arch of the aorta, and (as in the case of George I.) aneurism of that vessel. In all probability, though old narratives say the contrary, the heart's texture is never perfectly sound; even where the cause of the rupture has been external violence, some alteration of tissue has been generally found by recent observers.

III. In the majority of cases, rupture of the heart—itself complete and instantaneous—kills instantaneously. The hand is suddenly carried to the front of the chest; a piercing shriek uttered; some convulsive twitches occur, and the patient expires: or sudden loss of consciousness, from which recovery never takes place, marks the event.

There is a class of cases, too, in which, from the plugging of the fissure by coagula, the extravasation of blood into the pericardium is insufficient to stop the heart's action at once. Under these circumstances, a patient has been known to survive fourteen hours, with pallor, cardiac anguish, clammy sweats, coldness of the surface, feeble fluttering pulse, and sighing respiration; eventually going off quietly in a state of coma. The fact of death occurring so slowly suggests the question whether the permanent closure of a fissured opening does not fall within the range of the possible; and certainly one case of sudden death from rupture has been recorded, in which a former rupture was discovered, firmly filled by a fibrinous coagulum adherent to the wall of the heart.

A third class of cases exists, in which sudden cardiac anguish, attended with a sense of constriction, extreme dyspnœa, pain extending from the præcordial region to the left shoulder, coldness of the surface, giddiness and faintness, cramps, and small irregular pulse, are observed,—the whole series undergoing remission, nay, actually, it is said, totally disappearing temporarily, again to return with greater intensity than before, and close in death. In instances of this kind, in all probability, the heart's fibres have given way in successive layers.

IV. The greater number of cases have terminated fatally before medical attendance has been procured. Should the patient be still living, when first seen, such *treatment* should be employed as a common-sense view of the symptoms would suggest; for it is more than probable that the precise nature of what has occurred will rarely be diagnosticated with surety. Theoretically, the clear indication is to maintain the circulation in movement, with as little work on the part of the heart as possible; the head should be placed low, sedatives and slight stimulants administered, the surface kept artificially warm, and the very slightest movement of the body prevented. If reaction occurred, blood should be cautiously taken from the arm.

§ II.—I. Partial ruptures of the heart, or of its valvular apparatuses, are not extremely unfrequent. Rupture of the papillary muscles, permitting the ruptured ends to float, as it were, free in the cavity of the ventricle; rupture of the substance of the mitral or tricuspid valves, or of their tendinous cords; and of the pulmonary and aortic valves,—have all been observed *post mortem*, and their symptoms occasionally noted with more or less precision during life.

II. The immediate symptoms of the rupture of a chorda tendinea, especially if endocarditis have caused the accident, are not very prominent. The difficulty of the circulation through the heart, however, is increased; the pulse becomes very irregular, and symptoms precisely like those of the sudden formation of blood-concretions in the ventricles, make their appearance. The murmurs, constrictive and regurgitant, of the implicated valve ensue; but they may have pre-existed, as consequences of the endocarditis itself, and in that case positive diagnosis is an impossibility.

Sudden rupture of any number of cordæ tendineæ, or of a papillary muscle, especially when occurring independently of acute disease of the heart, produces very marked symptoms. Cardiac pain and anguish; palpitation irregular in force and rhythm; small, irregular, frequent pulse; syncopal tendency, overwhelming dyspnœa, dread of dissolution, pallor, coldness of surface, jactitation of the limbs,—all this suddenly occurring, in connection with a regurgitant systolic apex-murmur, which had been known not to have existed previously, might guide to the diagnosis, but not justify a positive assertion on the subject.

In a case of rupture of one of the aortic valves, observed by myself, symptoms such as those just described were attended with regurgitant basic murmur.

III. The character of the symptoms points to the propriety of administering stimulants and sedatives: sinapisms or the turpentine fomentation to the cardiac region relieve the anguish and constriction felt in that situation.

§ III. An individual, subject to palpitation, had a sudden attack of retching, with cardiac anguish, and died in an hour. The left coronary artery, widened, hardened, and fragile, was broken across from the aorta; effusion of blood had taken place between the pericardium and great vessels.*

Aneurism of the Heart.

§ I.—I. Aneurism of the heart occurs in two forms, corresponding to the fusiform dilated aneurism, and the lateral simple sacculated aneurism, of arteries (*vide* Diagram III., figs. 1. 3, p. 543): that is, a general and tolerably equable dilatation of a portion of the wall of a ventricle exists, or a pouched fulness rises abruptly (with or without constricted orifice) from the ventricle. Of either kind, aneurism is almost peculiar to the left ventricle. The compound sacculated aneurism of arteries, with injury to their walls (fig. 4), is also imitated by the heart, when destruction of the endocardium and, more or less extensively, of the nearest strata of muscular fibres precedes the pouching process.

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* Lombard; Gaz. Méd. de Paris, iii. 644.

In the majority of cases aneurism forms slowly, dependent, as it is, for existence on chronic changes in the substance of the heart; in some cases, however, its formation is an acute process, induced by ulceration or rupture of the endocardium and contiguous fibres.

II. Greatly more frequent (as 3 to 1) in males than females, —hitherto most commonly observed between the ages of twenty and thirty and in very advanced life, though few ages are actually exempt from the possibility of its occurrence,—having the ground-work laid for its formation in inflammation, fatty change, simple softening, or pseudo-fibrous infiltration of the heart's substance,—aneurism has been immediately traced, in a certain number of instances, to external injury, violent efforts, forced retention of the breath, and similar agencies, suddenly throwing an intense strain on the walls of the left ventricle. In the majority of cases its origin is slow and insidious—in fact, latent.

III. Under all circumstances the *symptoms* are obscure. When the onset is presumedly sudden, this has been announced by severe præcordial pain, orthopnœa, general agitation, dread of dissolution, syncopal tendency, and frequent, small, irregular, languid pulse. But obviously there is nothing distinctive in these symptoms: they indicate that the heart has received a deep shock of some kind, and nothing more.

On the other hand, where the disease is of slow origin and course, its symptoms do not seem to become serious, unless itself is carried to a great amount, or dilatation, with more or less hypertrophy, is added. The effects of such dilatation are then developed—systemic stagnation and its attendant evils in more or less complete array. At all events, it will be admitted that past records do not supply the materials of a clinical history of aneurism (as distinct from affections of the ventricles and of the orifices), however complete an anatomical one they have been made to yield by the zeal of Mr. Thurnam. Every known symptom of cardiac disease has been present, it is true, in these cases; but, as there either were positively, or may possibly have been, other morbid states present (I refer to narratives which make no affirmation as to the absence of such states), capable of causing the symptoms in question, it would be perfectly unjustifiable to ascribe them to aneurism.

And there is as great a dearth of knowledge of positive physical signs. The position of the heart's maximum impulse may perhaps be transferred from the apex to the base, where a sacculated aneurism springs from the neighbourhood of the base of the ventricle; probably, if the sac were prominent, the action of the heart would be attended with pericardial rub, not only systolic, but diastolic. Systolic murmur of blowing quality, of maximum force at the left apex, has been observed, undistinguishable from the murmur of mitral regurgitation. Though seemingly probable, *à priori*, that a double murmur might be produced by the ingress and egress of blood from the sac, where the orifice of this was narrow, experience shows that the diastolic portion may be completely wanting.* The signs of dilated hypertrophy frequently, of valvular disease sometimes, are coincidently met with.

IV. Death may occur suddenly from rupture of the sac into the pericardium, or, through an adherent pericardium, into the pleura: in the majority of instances the patient is slowly worn out with the symptoms of dilatation.

V. There is no special treatment for this affection: the symptoms and signs guide the physician to the adoption of the measures best adapted for hypertrophy or dilatation: the effects of systemic stagnation are to be averted or removed by the plans already described.

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* *E. g.* In Case V. of Dr. H. Douglas's Collection of Cases of Heart-disease, Edin. Monthly Journal, 1850.

§ II. Pouching, or aneurism, of the valves has occasionally been observed; always, as might be expected, in the direction of regurgitation, and produced by the force of the current playing on textures weakened in resistant power by chronic disease. This condition, anatomically known in the mitral, tricuspid, and aortic valves, has at present no clinical history.

POLYPOID CONCRETIONS.

I. Formation of fibrinous coagula within the heart during life is promoted by retardation of the circulation, occurring towards the close of exhausting diseases, in connection with weakness of the organ or mechanical obstruction to the circulation in its interior.* Hyperinosis carried to a great extent (as in pneumonia) has, on another principle, a somewhat similar tendency (p. 325). The condition of the blood in pyohæmia, and in other of the heteræmiæ likewise, favours coagulation within the heart; but endocarditis is, of all affections, its most frequent and effectual cause.

II. The symptoms and treatment of coagulative accumulation of blood within the heart in endocarditis have already (pp. 454-5) been described. In chronic diseases with languid circulation, the risk of coagulation should be averted as far as possible by abstaining from the use of nauseant medicines and digitalis (at least in any quantity), the obvious effect of which must be to slacken the circulation. Blood-letting should also be refrained from, unless under some urgent necessity. The influence of diluents (recommended for the purpose of rendering the blood less coagulable) can scarcely be trusted to,—and by producing a watery plethora they may do more harm than good. Small quantities of bicarbonate of potass, taken two or three times a day, seem to me the most promising prophylactic.

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* Hope says, he has seen many cases of phthisis in which coagulation of the blood occurred, from mere sluggishness of motion, in the femoral vein, with œdema of one or both extremities. I have never met with but one example of death from phthisis, with coagulation and obstruction of the veins in a lower extremity; in this instance, the femoral and iliac veins of the right limb were *inflamed* in the most positive manner (case of Henry James, U.C.H., Males, vol. v., p. 130, 1850). I cannot help doubting that Hope's observations can be frequently verified.

CHAPTER III.

DISEASES OF THE AORTA.

AORTIC PULSATION.

I. AORTIC pulsation, or abdominal or epigastric pulsation or palpitation, as it has been variously called, is a peculiar functional affection of the aorta, distinguished by more or less throbbing action of the vessel. Although very positively observable in the thoracic aorta, it is best known in the abdominal division of the vessel.

II. Pulsation at the epigastrium, more or less constant, but aggravated by various influences, such as brisk movement, nervous excitement, irritation of the bowels, or constipation and dyspeptic disturbance, is, as its title indicates, the prominent feature of the complaint. In rare instances, complete intermission of the throbbing action may occur,—a fact important in respect of diagnosis. When strongest, the pulsatile action is attended with a feeling of sickness or faintness, or pseudo-globus; there is no actual pain experienced, except the adjacent parts be accidentally extra-sensitive. The epigastrium, however, always bears pressure worse than in healthy people, and may be extremely tender.

In well-marked cases, pulsation may be easily seen at the epigastrium, especially in thin people; rarely at the umbilicus. The hand, laid on the surface in the course of the vessel, receives a forcible forward impulse, slapping rather than heaving, jerking, quick, abrupt, without distinctly expansile character, but bounding and free,—varying in degree from an action so trifling as to be scarcely perceptible, to one sufficiently powerful to shake the bed. As a rule, there is no lateral expansion to be felt; but, if hardened tissue lie on the confines of the vessel, such expansion may at the least be closely simulated. Unless the blood be anæmic, no thrill is to be caught. The vessel, if the patient be very thin, may be reached on either side by the fingers, and slightly moved laterally. The transverse limits of the vessel under percussion are natural; but it must be confessed this is a fact very difficult of establishment: the abdominal wall should first be steadily depressed for a minute or so, all gas and fæcal matters, as far as possible, pushed sidewards, and then the vessel carefully percussed. Aortic pulsation may exist in the highest degree without murmur,—a single systolic* impulsive sound being alone audible; or a systolic and a diastolic sound may be heard, the latter the fainter greatly of the two. Or, what is more common, a single systolic blowing murmur, prolonged slightly, rough and sharp, whiffing or whipping in quality, is heard,—the more marked, the greater the pressure exercised on the front of the vessel. Such murmur may, however, in rare instances be heard in the back. Until lately, I supposed that the discovery of double or of diastolic murmur, in the course of the aorta, positively indicated disease in its coats; but I now know, by a case observed with great interest during life, that there may be intense aortic pulsation, with diastolic murmur, though the vessel is perfectly free from disease, and in calibre below the average. In the instance referred to, however, the pancreas was found, after death, enlarged and hardened, and pressed somewhat on the vessel in the site of the murmur.†

Theory would lead to the idea, that in cases of pulsation limited to the aorta, that vessel should be the seat of some

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[*] In applying the terms systolic and diastolic to mark the rhythm of sounds and murmurs in the arteries, I intend to signify synchronism with the systole and diastole of *the heart*, unless the contrary be stated expressly.

[†] Case of Eliz. Gosling, U. C. H., Females, vol. v., p. 130.

organic change; else why such limitation? Few examples are recorded of *post-mortem* investigation; in some of the number, affirmation is absolutely made that the vessel was in all respects sound; in others, that its walls were flaccid (but as cause or effect of the pulsation?); in yet others, thin. The calibre of the vessel may be reduced by the pressure of adjacent morbid structure. However produced, the effects of aortic pulsation are sufficiently serious: it increases already existing nervousness, excites apprehensions on the part of the patient of deep-seated organic disease, deprives him sometimes of the power of taking exercise, interferes with digestion (whence loss of appetite and emaciation); and some patients, constantly dwelling on the symptom, magnifying its importance, and fancying that the by-standers must notice it as well as themselves, acquire an utter distaste for all society. The duration of aortic pulsation may be very considerable; Baillie thought that once developed, though it might vary in amount, it seldom disappeared altogether: where produced by anæmia, it is positively, however, susceptible of complete cure.

Aortic pulsation is not so common in ordinary hysteria and spinal irritation as might be expected; it accompanies many utero-ovarian diseases, with pelvic and abdominal neuralgiæ; follows anæmia of all modes of origin; occasionally attends plethora, sthenic and asthenic; and is induced in some susceptible frames by green tea, strong coffee, tobacco, and similar agents. Various disordered states of the chylopoietic viscera produce it—simple dyspnœa, flatulence, and hepatic disturbances among the number; but special proneness to the complaint probably exists where causes so slight suffice for its generation. Acute inflammation of the stomach, bowels, and peritonæum, as insisted on by Dr. Stokes, and chronic gastritis, as chiefly illustrated by Dr. Faussett, sometimes produce very marked sympathetic action of the abdominal aorta. Pressure of all kinds directly on the vessel takes an important place among its causes; I have known an accumulation of fæces in the transverse colon induce it in an aggravated form.

III. A very little care will distinguish pulsation of the aorta in the epigastrium, from the epigastric pulsation of a displaced heart, or an enlarged right ventricle; it is unnecessary to dwell on their distinctive marks. But there may be great difficulty in the diagnosis between functional and aneurismal pulsation of the vessel. In the latter case, however, the pulsation is expansile, heavy, powerful, slow, and appears kept back, as it were, by some restraint behind it, instead of bounding freely forwards; thrill is occasionally to be felt, and a tumor, with dulness under percussion nearly commensurate with its size, is discovered. A harsh, grating, hollow, murmur, systolic, rarely diastolic, sometimes double, audible in front, may also sometimes be detected along the spine posteriorly. All this is accompanied with pain more or less tearing or agonising; and the distinction of the cases is not difficult.

But where an aorta, healthy in itself, is pushed forwards by an enlarged vertebra, or a tumor connected with the spine,—or where an indurated mass lies in front of, or to either, or both, sides, of the vessel, lateral expansion may be simulated, the murmur may be harsh, though not grating,—while local pain and general emaciation may be produced by the organic morbid state, whatever it is, superadded to the pulsation. Under these circumstances, the difficulty of diagnosis may be extreme. It appears from the case just referred to, that even the discovery of diastolic murmur in the vessel (that is, synchronous with its own systole) will not prove the existence of aneurism positively; still such murmur very very rarely attends inorganic pulsation. As a rule, too, the murmur of the latter origin is inaudible in the back; but that of aneurism may be similarly limited in extent: the sex of the patient may give accidental aid; for aneurism of the abdominal aorta is very rare in the female, aortic pulsation common. Again, the existence of inorganic murmur in the heart, thoracic aorta, and veins may assist also; but it may tend to deceive too, for the subject of aneurism is not exempted from becoming anæmic. In ordinary cases, then, the diagnosis is easy; in some rare instances it will be well to watch the case for a time before risking an opinion, and to positively affirm the absence or presence of slight peripheral dilatation of the coats of the vessel (Diag. III., fig. 1, p. 543), may, even after prolonged observation of the case, remain impossible.

IV. In the majority of cases, the *treatment* of aortic pulsation directs itself to the removal of its causes,—such as spanæmia, spinal irritation, gastric or intestinal disturbance, lucorrhœa, &c.

When the affection is more purely nervous, its management should be conducted as follows:—all suspected articles of food, strong tea, and coffee, and similar stimulants, should be forbidden, and a plain, nutritious diet rigidly adopted, with but little vegetable or other substances that promote flatulence. An occasional warm aperient, with some carminative adjunct, is advisable. Antispasmodic medicines afford relief to the symptom, though they fail to reach its cause; of these valerian, æther, ammonia, asafœtida, and musk may be employed; sedatives, such as lettuce, conium, and hyoscyamus, may be given in alternation with others of the class acting more especially on the heart, namely aconite, digitalis, and hydrocyanic acid. These medicines are beneficially combined with tonics, where the general indications for such agents present themselves.

The application of a few leeches to the epigastrium (four or five) sometimes distinctly tranquillises the pulsating vessel; even where no fair suspicion exists of the presence of gastritis in any form. Dry-cupping, a fact not easily explained, I have sometimes found beneficial; anodyne embrocations, belladonna plaster, and the endermic use of morphia, in the scrobiculus cordis, moderate the arterial action.

Change of air and travel, moderate exercise, daily friction of the skin, the shower-bath, sea-bathing, or the tepid salt-water bath, and, in fact, all hygienic influences, that strengthen the nervous system and improve the health generally, are among the most effectual agents in the cure of obstinate cases.

Aortitis.

I. *Acute aortitis* is a rare disease, at least as far as demonstration of its existence goes; it appears to be singularly uncommon, where it might be frequently expected, namely, in connection with acute endocarditis.

II. The signs of this affection are obscure,—at least in the present state of knowledge. Violent pulsation of the vessel, and tumultuous action of the heart, sometimes exist; it is very probable that, if lymph be deposited to any extent on the lining membrane, thrill may be perceived, where the vessel nears the surface of the chest. In a remarkable case, observed by Dr. Parkes,* an extremely loud, rough systolic murmur continued audible from the third dorsal vertebra quite down to the lumbar region—a murmur obviously due to the passage of the blood in the vessel over a surface roughened by patches of lymph. In this instance the pulse was irregular and small, but the aortic orifice was contracted, and otherwise diseased, and the heart in a state of dilated hypertrophy; it does not appear that in the uncomplicated inflammation the pulse becomes irregular.

Intense general uneasiness and jactitation are very usual symptoms; general tenderness of the skin has been noticed by Dr. Bright; rigors announce the onset of the disease; and M. Bizot insists much on the significance of general acute œdema of the trunk, arms, legs, and face. Dyspnœa, it appears, may be absent, when the disease is simple; yet it is difficult to conceive that the vessel can be inflamed to any extent without affecting the respiration: generally speaking, other affections, directly implicating the action of the lungs, co-exist. From some observations by Dr. Corrigan, it would appear that inflammation of the mouth of the aorta may induce a series of pseudo-anginal symptoms; but, on the other hand, that such is not a necessary effect is shown by Dr. Parkes's case. Pain, with sensation of heat, in the course of the vessel, complained of inferiorly (both anteriorly and posteriorly, on the level of the lumbar spine), has occasionally been a prominent feature. Syncopal tendency and apprehension of immediate death were noticed by Dr. Corrigan. Of the state of the urine nothing is known; yet, as we shall presently see, this is probably a matter of considerable importance.

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* Medical Times, Feb. 23, 1850.

Dr. Chevers, who has given much attention to the diseases of the vessels, infers from collated cases, that death occurs in acute aortitis with extreme prostration, sharpened or bloated and livid features, cold and discoloured surface, rapid indistinct pulse, stertorous respiration, swollen extremities, and duskiness of the superficial veins; the patients die comatose, and altogether with the aspect of persons destroyed by an animal poison. There is an asthenic variety of the disease, too, in which the symptoms are adynamic from the outset.

III. It will, I think, be generally conceded that the elements of a positive diagnosis of acute aortitis are yet to be found. If narratives may be implicitly trusted to, cases occur where, supervening on other affections, acute or chronic, acute aortitis produces no obvious effect except increased irritability and distress: clearly, the diagnosis of the disease could not, under such circumstances, be ventured on. Pain, thrill, and pulsation in the course of the vessel, with arterial murmur coasting the spine, and answering in localisation neither to a murmur of the aortic nor of the mitral valves, would be the conditions, coupled with great general distress and febrile action, most nearly warranting the diagnosis of the disease. But it is needless to point out the varieties of states that might simulate the entire series, except the aortic murmur; and in respect of this murmur, the possibility of its depending on chronic disease points to the necessity of caution.

According to M. Bizot, œdema of the trunk, arms, face, and lower extremities, occurring acutely, without functional disturbance of any organs but those of the circulation, indicates acute aortitis. But, on the one hand, Dr. Parkes's case (one of the most indubitable on record in regard of anatomical characters) proves absolutely that the disease may exist in perfection without any such œdema; and, on the other hand, M. Bizot has not taken into consideration the state of the kidneys in his cases. Now, in one of the three, Bright's disease appears to have been positively present; and in the other two we have no assurance that it was absent. M. Bizot's observations were made before renal diseases were either clinically or anatomically paid much attention to.

IV. Aortitis may prove very rapidly fatal; it has certainly destroyed life in three or four days in association with other less serious states. The transparence of exuded lymph furnishes the best measure of the recency of the disease. Of the prognosis, all that can be said is, that as persons die of various diseases with the evidences of chronic inflammation in the vessel, either acute aortitis sometimes fails to kill, or chronic aortitis sometimes pursues a chronic course from the outset: both propositions are probably occasionally true.

V. Were the disease upon fair grounds even suspected, active measures should without delay be had recourse to. Venesection or free cupping in the course of the vessel in the front of the chest and along the spine, or if the disease have occurred in a very low state of system, dry-cupping, are clearly indicated. Counter-irritation, by the application of a long narrow blister along the left vertebral groove (which may be used also for the purpose of applying calomel and morphia endermically), has theoretical argument in its favour; for the mass of tissues, intervening between the skin and vessel, is in all probability sufficiently great to render the effect of the blister *antagonistic*.

The internal medicines, deserving of most confidence, are calomel, opium, and tartarised antimony: if the excitement of the heart and vessel were very great, digitalis or acetate of lead might be simultaneously administered. Aperients and saline diaphoretics are worthy of attention as adjuvants.

In employing these and other measures the practitioner must never lose sight of the constitutional state.

VI. *Chronic Aortitis.*—Thickening of the coats of the aorta, undue vascularity of the outer membrane, unevenness, roughness, puckering, furrowing, and channelling of the inner surface of the vessel, with various alterations in its calibre, producing irregular distension and contraction, constitute unquestioned characters of chronic inflammation. White opaque cartilaginiform patches, studding the inner surface of the vessel, are likewise admitted to be the chronic representatives of the lymph-exudations of the acute disease. The relationship of saline precipitation to these patches is made matter of dispute; the fact appearing to be, that, while the usual nidus of calcification is certainly atheroma, the white patch is the occasional seat of the change, either primarily or secondarily to the deposit of atheroma within itself.

But, serious as these conditions are anatomically, we know of no symptoms to which they give rise, unless they have led to (or, at least, are attended by) very considerable alterations of calibre of the aorta, in the forms of obstruction, coarctation, or dilatation,—conditions which will presently be more fully considered. Nor is it readily conceivable, indeed, *à priori*, that much local disturbance should be produced by these changes. Pain of a notable kind can scarcely be expected; and until the elasticity of the vessel has been very deeply impaired, it continues capable of taking its part—in great measure a mechanical one—in carrying on the circulation.

ATHEROMA AND CALCIFICATION.

Atheroma and calcification of the aorta, conditions of great anatomical interest, have in themselves but little clinical importance. Destroying the elasticity of the vessel, rendering it fragile, contributing to the production of various aneurismal changes, and connected with two important diatheses—the fatty and the calcifying—the prominence of these conditions of the vessel can scarcely be overrated in a pathological point of view: and in their ultimate possible influence they are of grave clinical interest; for calcareous matter may be the occasion of obliteration of the vessel, by protruding more or less into the interior, and affording points for the blood to coagulate around. Yet, until such obstruction more or less prominently displays itself, what symptomatic indication have we of atheromatous or fatty deposition in the coats of the aorta? None, certainly, in the present state of knowledge. And of physical evidences there are none, except systolic rough murmur in the course of the vessel, either limited thereto, which is rare, or audible in a less intense form at the aortic valves, also (p. 245),—jerking, inelastic impulse behind the sternal notch, and, occasionally, systolic thrill both at that point and about the second right cartilage.

COARCTATION AND OBLITERATION.

I. The arch of the aorta is subject to a peculiar *quasi-congenital* coarctation, which occurs at the time of the closure of the ductus arteriosus, is connected immediately in position with that duct, may pass on to total obliteration, in the great majority of cases produces but little ill effect, and has never, so far as I know, been positively diagnosticated during life.

In some cases symptoms resembling those of aneurism of the arch have been noted, dyspnœa, palpitation, and pain behind the sternum: œdema of the ankles does not appear among those mentioned, unless where enlargement of the heart or valvular disease co-existed. Violent throbbing of the carotid and temporal arteries, an occasional symptom, is explicable by the enlarged calibre of those vessels.*

Loud systolic murmur, of maximum force at the second right cartilage, but greatly more intense at the upper part of the sternum generally than at the cardiac region (where it is evidently audible only by transmission), accompanied with well-marked thrill in the second interspace on each side of the sternum, and also above this bone, may be expected (judging from two cases reported by Dr. Blackiston) to attend this coarctation of the vessel. The amount of thrill will probably vary with the precise position of the arch, the condition of its inner surface, and the state of the blood. Highly-marked aortic systolic murmur and thrill will not then justify positively the inference of aneurism: some of the other physical signs of the latter disease, such as local prominence and dulness under percussion, or some of the class of concentric pressure-signs, are required in addition to warrant its diagnosis. Anæmic blood, rushing past the inequalities of surface, caused by irregular calcification of the arch, might give rise to a murmur limited in situation to that portion of the vessel.

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* Nothing can be more curious than the anastomotic enlargements that occur in these cases. Carswell's Drawings, U. C. Museum, A. 20, exemplify this: the innominate artery is considerably thicker than the arch, the arteries of the base of the neck greatly enlarged, and nameless ramusculi as full as the radial artery in the natural state; the internal mammary and epigastric arteries are especially of large calibre.

If, then, an aortic murmur, barely audible at the heart, were discovered as a permanent condition in a person free from spanæmia, and if there were neither undue dulness under percussion nor bulging of the surface in its site, and the patient suffered neither from the local or general symptoms, nor the concentric pressure-signs of aneurism or tumor, there would be fair motive for strongly suspecting the existence of coarctation of the arch. This view would be greatly strengthened if the right carotid and sub-clavian arteries were obviously enlarged and prone to violent throbbing; otherwise all the conditions enumerated might depend on an obstruction at the aortic orifice of the innominate artery.

The possibility of living on in excellent health with a coarctation of this kind is amply proved by recorded cases. Adult, nay aged, patients, have been cut off by various diseases in whom it had existed to a very high degree without having ever attracted attention during life to the organs of circulation: its existence has, in truth, been wholly a *post-mortem* discovery. On the other hand, violent efforts, by over-filling and straining the thoracic organs of circulation, *may* at any time cause sudden death. Under such circumstances, rupture of the aorta on either side of the ductus arteriosus, and rupture of the left ventricle and right auricle have been observed.*

II. The calibre of the aorta may undergo diminution of an *acquired* kind, as a result both of its own diseases, of certain diseases of the heart, of the lungs, and of others of the system at large. Under the first head appear contractions depending on exudation-matter connected with the coats of the vessel. Under the second, range themselves reductions of size depending on the closely constrictive action of induration-matter in the pericardium and obstruction of the mitral orifice, which entails a deficient supply of blood to the aorta (p. 500). Next, prolonged obstruction of the pulmonary circulation acts in a somewhat similar way: the calibre of the aorta is below the average in persons cut off with vesicular emphysema of long duration, unless the right ventricle have become the seat of eccentric hypertrophy. Lastly, in cancer and phthisis the vessel suffers in the same manner, probably from the gradual reduction in the amount of circulating fluid in those diseases.

III. A considerable number of cases are on record in which imperviousness, more or less absolute, of the aorta has been produced in the adult by coagula forming around prominent spiculæ of calcification, or by the products of inflammation, aided by contraction of the walls of the vessel itself. The lower part of the thoracic and the abdominal aorta are the most common seats of the disease.

The course of this affection, symptomatically, may be acute or chronic. In the former class of cases, doubtless, the current of blood, though it may long have been somewhat obstructed, has not become seriously interfered with until the appearance of acute symptoms,—these symptoms being dyspnœa, anasarca, tendency to gangrene of the lower extremities, and hæmoptysis. But as the state of the lungs and heart is imperfectly known in the very few instances of the kind, the direct dependence of hæmoptysis on the obstruction may be questioned.

In 1835, I saw in the wards of M. Louis a remarkable example of obliteration of the abdominal aorta, in a female aged fifty-one. Here, four years previous to death, numbness, first of the right, and, some months later, of the left, lower extremity, were clearly the earliest positive effects of the growing obstruction. These were followed by inability to walk, not from fatigue or cardiac suffering, but from pain, coldness, and increasing numbness in the legs. Subsequently, the effects of organic disease of the heart (she had mitral constriction, dilated hypertrophy of the right ventricle, palpitation, pulmonary apoplexy, and hæmoptysis) threw those of the aortic obstruction into the shade, and eventually cut her off in a few days after admission to the hospital. Twice in the course of her illness she had slight and passing œdema of the lower extremities, but there was *none* on her admission, and at death scarcely any. Besides the local obliteration, the calibre of the aorta generally, and of its branches, was below the average in this woman. No attempt appeared to have been made at anastomotic enlargements; and the disease had, to all seeming, originated in inflammation of the vessels.

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* *Vide* Crisp, Dis. of Blood Vessels, p. 31.

Aneurism.

I. The term "aneurism," understood in its widest sense, may be defined as a local increase of calibre of an artery. And in this sense it has been used by some authors, while others have made attempts to restrict its application in many different ways. Professional opinion is, indeed, so unsettled as to the proper application of the term, that an explanation of the sense he, in particular, may attach to it, is called for on the part of every person employing it.

Adhering, then, to the comprehensive definition above expressed, I would divide the *genus* Aneurism into the subjoined *species* and *varieties*:—

A. Peripheric: *dilating*	Fusiform. Globular.
B. Lateral: *sacculating*	a. Simple. b. Compound. c. Mixed.
C. Interstitial: *dissecting.*	

The anatomical constitution of these varieties of aneurism, is exhibited to the eye in Diagram III.

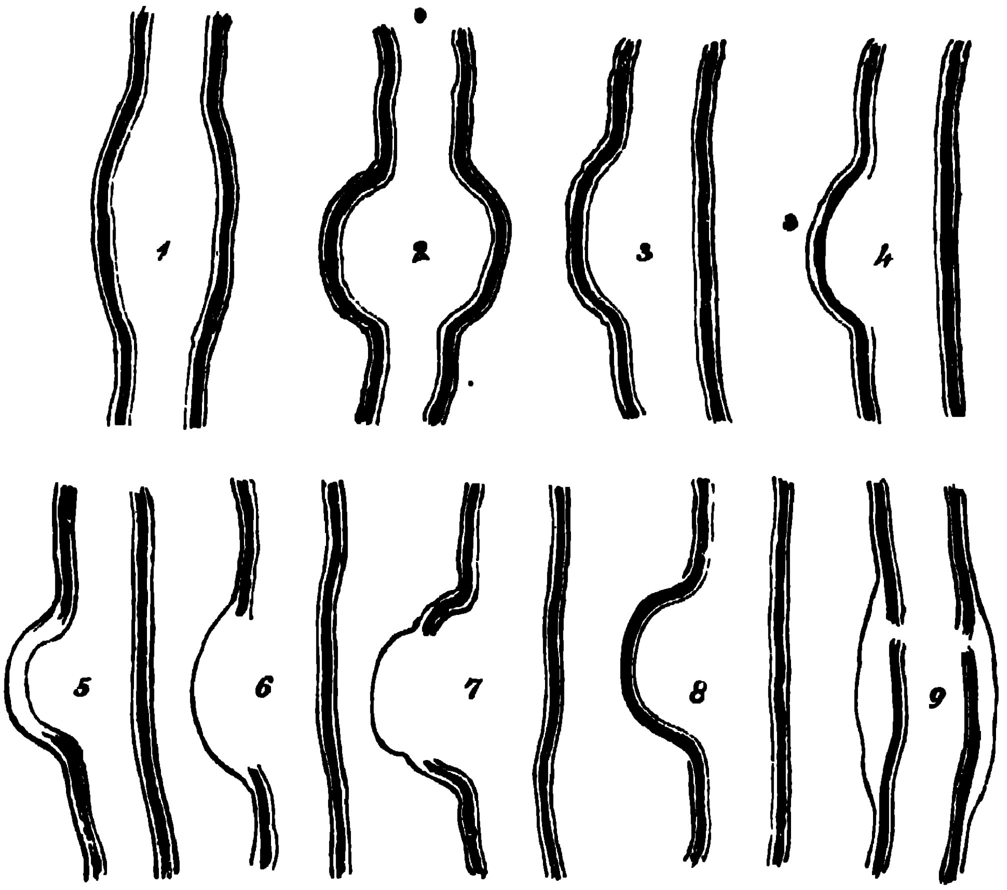

Fig. 1, Peripheric Dilating Aneurism; the three coats of the vessel all round *gradually* widening, so as to give a *fusiform* shape to the enlargement. Fig. 2, Peripheric Dilating Aneurism; the three coats of the vessel all round *abruptly* widening, so as to give a *globular* shape to the enlargement. Nos. 1 and 2 are the "simple dilatations" of various authors. Fig. 3, *Simple Lateral* Sacculating Aneurism; the sac, formed of the three coats of the vessel unbroken, rises from a limited portion of its circumference: the "*true* aneurism" of some authors. Figs. 4, 5, 6, 8, varieties of *Compound* Sacculating Aneurism; all agreeing in the characters of lateral sacculation, and injury to the coats of the vessel: the "false aneurism" of writers. Fig. 4, the Sac formed of the middle and outer coats, the inner being destroyed. Fig. 5, the Sac composed of the outer coat lined by the inner, the middle tunic having disappeared. Fig. 6, the Sac, composed of the outer membrane alone. Fig. 8, the Sac, composed of the middle and inner coats, forming a sort of hernial protrusion through the destroyed outer coat. Fig. 7, Mixed Aneurism, a combination of the simple and compound sacculating varieties; the simple condition of Fig. 3 having existed for a variable time, the middle and inner coats more or less suddenly give way, and the condition exhibited in Fig. 6, is added to the original simple disease. Fig. 9, Dissecting Aneurism; separation of the outer coat from the middle, by blood escaping from the interior of the vessel, through fissures in the lining and middle tunics.

Now, in the aorta, the only vessel with which we have here to do, certain of the above varieties are distinguishable by symptomatic characters, and, in reference to this important practical fact, may be placed in four *clinical* groups, thus:—(*a*) the simple and compound lateral sacculating aneurism of figs. 3, 4, and 6; (*b*) the fusiform dilating aneurism of fig. 1;* (*c*) the mixed aneurism of fig. 7; (*d*) the dissecting aneurism of fig. 9.† By far the most important of these groups is the first. Its constituent varieties in the present state of knowledge are not distinguishable by any positive signs during life; hence their association in this clinical scheme.

The effects, signs, and characters of aneurisms of the aorta differ so much in the chief divisions of the vessel, that the separate consideration of the disease in the arch (with the ascending aorta conjoined), in the descending thoracic and in the abdominal divisions, is matter of absolute necessity.

ANEURISMS OF THE ARCH OF THE AORTA.

GROUP *a.*—*Simple and Compound Lateral Sacculating Aneurisms.*—I. Affecting a portion only of the circumference of the vessel; narrower generally (the compound much more so than the simple) at the neck than at the body of the sac; rarely springing from the inferior curvature of the arch; ranging in size from that of a nut to that of the fœtal head; filled to variable extents with laminated coagula (the compound more extensively, as a rule, than the simple, because the orifice of its sac is comparatively narrow); the influences of these aneurisms must seriously differ, according to the precise direction in which the sac chances to enlarge. The grand practical distinction of aneurisms, in this point of view, is into the *eccentric* and the *concentric*,—those tending to grow outwards towards the walls of the chest, or inwards towards deeply-seated parts. The former habitually attain much greater bulk than the latter, for the simple reason that organs of vital importance being secure from pressure, the patient suffers comparatively little, life is prolonged, and time given for the free enlargement of the sac.

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* Globular dilating aneurism I have never seen in the aorta; it appears to be most common in the cerebral arteries.

† I strongly doubt that the sac, in Fig. 5, is such as it has been represented to be by authors,—namely, the outer coat lined by the stretched inner membrane. The apparent inner tunic is probably nothing more than a film of adventitious formation; at all events, I have never seen a sac so constituted. Of Fig. 8, there is but one positive example known, so far as I can ascertain,—namely, the preparation, No. 1642, in the Hunterian Museum. Another variety of hernial aneurism (protrusion of the inner through the middle and outer coats) has been described: in all probability, imaginatively.

The effects of an aneurismal sac on the adjacent parts are of various kinds; but all, directly or indirectly, due to pressure. Mere *displacement* is illustrated by detrusion of the trachea, or œsophagus, sidewards or backwards, of the heart downwards; by anterior bulging of the walls of the chest; by dislocation of the sternal end of the clavicle; and by depression of the apex of the lung.* *Interference with the freedom of hollow canals* is seen in obstruction of the œsophagus, the trachea, either large bronchus, the descending cava, the innominate, left carotid, or left subclavian arteries,—vessels which, blocked up either by direct pressure, by twisting of their orifices, or by coagula, become more or less functionally disabled. *Injury to parenchymenta* is exhibited in condensation of lung-substance, condensed so as to be incapable of expansion. *Destructive absorption of tissue* may occur in the trachea, the œsophagus, the pleura, lung, and bronchi; the pericardium; the pulmonary artery; the substance of the lung itself; the thoracic duct; the ribs, sternum, vertebræ (aneurism of the arch rarely, however, actually makes its way into the spinal canal); the recurrent nerve, and the spinal nerves on the left side. *Exudation of lymph* often takes place between the aneurismal sac and the parts pressed on, as the ribs and sternum, the pleura and lung. *Irritation of nerves* is exhibited in various spasmodic phenomena connected with traction of the recurrent nerve. And, lastly, actual *inflammation of texture*, as an indirect result of pressure, is displayed in the tracheitis and bronchitis that so often accompany the disease. Now, as will be readily understood, many of these different effects are clinically useful in guiding the observer to a precise localisation of the disease.

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* Case of Brader, Clin. Lect., loc. cit. No lung-substance reached higher, in this instance, (aneurism of descending portion of the arch), than the second rib on the affected side.

II. The physical signs of a sacculated aneurism of the arch are in well-marked cases extremely numerous. (*a*) By inspection, local bulging is discovered, tending, when at all notably prominent, to the gently conical form. The ribs and interspaces equally contribute to its formation, when the area of the bulged surface is at all extensive; but when limited (that is, at the time the sac commences first to act on the surface), a single rib or cartilage may alone be prominent, or the end of the clavicle simply pushed forwards. In cases of long standing, and where the base of the conical bulging is extensive, the skin becomes smooth and glazed-looking, the unevenness of the ribs and interspaces being completely removed. Concerning the seat of this bulging: if the sac spring from the first division of the arch (including what is called the ascending aorta), the prominence mainly appears at the right edge of the sternum, in the second interspace and second cartilage, up to the first, and even as far sometimes as the clavicular joint. But if, while the sac involves this portion of the arch, it also implicates the transverse portion and somewhat the descending also, the prominence, instead of lying to the right, may be placed solely to the left of the sternum, in the infra-clavicular and mammary regions, extending as far outwards as the line of the nipple,* obviously the ascending part of the arch gets twisted to the left. Secondly, if the transverse division be the affected part, the top of the sternum, or the right

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* Case of Harris, U. C. H., Males, vol. ii., p. 262. The sac in this remarkable case commenced two inches above the aortic valves, and terminated close to the left subclavian; the innominate artery was carried in front of the origin of the left carotid.

first cartilage form the prominence; but if the sac be of small size, actually limited to this segment of the vessel, and springing from its posterior aspect, there may be, or, more correctly, will be, no visible bulging at all. Thirdly, when the descending portion of the arch is sacculated, the prominence appears at and about the second left cartilage: but aneurisms thus situated do not often cause anterior bulging; the vessel is here far away from the front of the chest, and, as matter of experience, the tendency of its aneurisms is to enlarge to the left side, while they expend their force posteriorly in eroding the vertebræ. Whatever be its seat, the superficial extent of the prominence is less than the diameter of the sac; the bulged surface corresponds to the most prominent part of the latter. The actual area varies between the size of half-a-crown and that of a large cocoa-nut. Deficiency of bulging is rarest where the sac lies to the right; commonest, where it lies to the left; of medium rarity, where the horizontal and central portion of the arch is implicated.

Movement, pulsatile, expansile,* and synchronous with the heart's systole, may be seen in the bulged part of the chest; and, in some cases, motion of more abrupt and non-expansile character is very perceptible to the eye above the clavicles, evidently coming from below and as evidently produced by a mass of some bulk. But if a sac, even though of massy bulk, be filled in great measure with fibrine, while a dwindled amount of blood trickles with more or less difficulty through a small channel at the furthest aspect from the external surface, it may, especially if seated in the descending part of the arch, be as absolutely pulseless to the hand as if totally unconnected with the arterial system.

(*b*) The hand, applied to the bulged surface, appreciates more accurately the motion of the sac,—generally a very little behind the apex-beat of the heart in point of time, its systolic impulse is sometimes, as far as the sense of touch can determine, synchronous with this. By systolic impulse we mean that synchronous with the systole of the heart: if the vessel alone were considered, this impulse would of course be called diastolic. In certain instances, by no means in all, the impulse is double, a receding as well as an expanding motion existing. The force of the systolic impulse is sometimes extreme; even from a sac of small size, and mainly seated behind the sternum (which has undergone but very slight erosion) it may be sufficiently strong to shake the head applied to the stethoscope.* In character, throbbing simply or thrusting and heaving, dull and inelastic (the latter condition marked in proportion to the quantity of fibrine in the sac), there are cases in which it is very positively undulatory. When the walls of the sac reach directly to the surface,—when they have bulged this considerably,—when there is a free passage through the sac,—and when laminated coagula in but small quantity lie in its interior, the current of the blood passes with wave-like motion directly beneath the finger laid on the skin. The seat of motion, as felt, is the same as of the visible action. When the descending portion of the arch is aneurismal, the impulse of its transverse division, which we will suppose sound, may be considerably intensified, as felt by a finger placed in the sternal notch; or it may vary from day to day, independently of any concomitant change in the impulse of the prominence directly over the sac,—a circumstance sufficiently calculated to puzzle the observer as to the real seat of the disease. Aneurisms seated in this part of the arch tend also to raise its transverse division slightly above the natural level. In rare instances, very positive impulse may be detected in the inter-scapular region, more frequently on the left side than the right,—a fact explained by the relationship of the arch on the two sides to the spinal column. Systolic vibratile thrill of variable amount may attend the impulse, or be perceptible even where distinct impulse cannot be detected. In aneurisms of the group under consideration, thrill is less constant than in the group of peripheric dilatations; but I have known it intensely marked, sufficiently so almost to tickle the hand. Thrill may be limited to the bulged surface, extend slightly in all directions beyond its confines, or be perceptible above the clavicles and sternal notch. The sign may be more distinct in the latter situation than over the sac itself. Thrill may exist at one time, disappear and return more forcibly than ever,—a fact in some instances explicable by the varying state of the blood in respect of anæmia, in others referrible to changes in the state of the sac, its contents, and its inlet.*

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* The expansile character of the pulsation was shown to the eye remarkably in the case of Warren, U. C. H., Males Ward 4, October 22nd, 1849. A belladonna plaster had been placed over the aneurismal bulging, which was remarkably broad-based; as the plaster produced an uncomfortable feeling of tension at first, it was slit across horizontally; subsequently, with each throb of the sac, the edges of the incision diverged by about a line.

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* Case of Downie, U. C. H., Males, vol. v., p. 273.

(*c*) Aneurismal sacs, according to the direction in which they point, increase the measured distance from the sternal notch to the nipple, the middle line to the nipple, or both. They interfere with the measurable chest-expansion of breathing on the surface corresponding to themselves, and may restrain this very greatly by pressure on a chief bronchus.

(*d*) Dulness, with resistance, under percussion, exists in the surface nearest an aneurismal sac. The reasons why the superficial extent of dulness should prove, practically speaking, less than that of the aneurismal pouch and artery, from which it springs, combined, have already been explained (p. 241). An area of some square inches may be rendered dull by sacs of very large dimensions. The observer must always bear in mind the possibility also of the real dulness of a sac being factitiously increased in extent by adjacent consolidated lung, or solid accumulation in the mediastinum.

The seat of dulness in *front* of the chest bears the same relationship, as that of prominence, to the different portions of the arch. As a rule, it is most easily and completely detected to the right of the sternum and in connection with the ascending portion of the arch and its angle on the right side; but if twisting of the vessel take place, the deficiency of resonance is most obvious to the left of that bone. Nor can it be said that a sac springing from the descending portion of the arch, even when small, fails to effect the percussion-sound; but, for obvious topographical reasons, it does so proportionally less, both in superficial extent and in amount. A sac originating in the horizontal part of the arch impairs the natural osteal clearness of the resonance immediately below the sternal notch, the edges of the lungs being pushed sidewards to a variable extent: it also affects the sound above and to either side of the bone. Whatever be the seat of the sac, it is important to ascertain positively whether the dulness connected with it does, or does not, reach completely into the acromial angle of the infra-clavicular region. Aneurisms of moderate size seated in the horizontal and descending parts of the arch give dull sound *posteriorly* in the inter-scapular regions; and a large sac, even though derived from its ascending portion, will impair the resonance between the right scapula and the spine.

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* *E. g.* Case of Harris, loc. cit.—Thrill disappeared Dec. 4, returned Dec. 21.

If a sac be moderately stratified internally with fibrine, the character of its resistance is in nowise special. If, on the contrary, it be closely filled with such coagula, and the amount of fluid blood within it be small, the resistance is dull, inelastic, and putty-like. This kind of resistance is significant enough, when discovered; but it is scarcely necessary to add, that, either for the purpose of eliciting this, or any other, character of percussion, the least roughness in manipulation is not only awkward and *unclinical*, but actually dangerous.

In a former chapter (p. 242) I have touched on the question of the smallest amount of dilatation of the arch capable of being demonstrated by percussion. Under a concourse of favouring circumstances, we have seen that a very small increase of size may be so discovered. The inefficiency of percussion increases with the smallness of the sac and its distance to the left of the median-line. There can be little doubt that more than one small aneurismal sac, in the middle division of the arch, has escaped detection, simply because percussion was not performed below, and at, the sternal notch.

(*e*) Few diseased states give rise to such variable auscultatory signs, as a sacculated aneurism of the arch,—a fact sufficiently proved by the following list of conditions of sound that have actually fallen under my notice: the list probably might be increased from the experience of others. Through the stethoscope, placed on the most prominent part of the surface, may be heard—1. A systolic blowing murmur, harsh in quality, yet less so than elsewhere in the artery close to the sac; with a dull, muffled diastolic sound. 2. A double sound, both divisions of which are rendered murmurish by suspension of the respiration, and both are weaker than the sounds at the base of the heart. 3. A double sound of the same characters, except that its divisions are louder than the basic sounds of the heart. 4. A roaring, grating systolic murmur, stronger than at the base of the heart, the aortic valves being constrictively diseased, and the blood spanæmic; with a diastolic sound. 5. A double rough murmur, the systolic division louder than the diastolic. 6. A double rough murmur, the diastolic division louder than the systolic. 7. A systolic sound, with a diastolic murmur (very rare). 8. No sound at all, properly speaking; but a dull, impulsive impression (systolic), that simulates sound. Occasionally, a peculiar character in the single or double sound of an aneurismal sac, when completely free from murmurishness, may be perceived, that seems best designated by the phrases *pumping* or *sucking*.

Of the characters of aneurismal murmurs, the following are worthy of clinical attention. The quality of the *systolic* murmur may be simply blowing, or blowing with a peculiar hoarse hollowness (which, when well marked, is important in diagnosis), grating, rasping, sawing, filing, or, if the blood be spanæmic, roaring. Variable, but generally low, in pitch, this murmur may be of higher pitch, nevertheless, than a co-existent systolic murmur at the base of the heart. Short and abrupt in the majority, prolonged, almost drawling, in the minority of cases, its intensity may reach, or even exceed, that of the loudest murmurs produced within the heart. The strength of the *diastolic* murmur, though variable, is rarely great, absolutely speaking; in quality, it is generally softer than the systolic; it is not constantly present, not only at different periods of the same case, but even with successive beats of the heart. Generally best audible over the most projecting point of the prominence, murmurs may, on the contrary, attain their maximum loudness at the edges of this; or (even when the main part of the aneurism lies considerably below the clavicles), at the base of the neck,—probably, under the latter circumstances, thick layers of fibrine are accumulated in the lower parts of the sac; or, lastly, (this is very rare) on the left side of the spinal column.

The mechanism of these murmurs seems to be this. The systolic is either produced by the passage of blood over a surface roughened by fibrinous masses, inequalities in the coats of the vessel, and calcifications; or it may come of the rippling motion given to the fluid on its entry from a portion of tube of natural calibre into one more or less dilated, especially if the dilatation be abrupt; or it may be caused by the flow of blood through the comparatively narrow and more or less rough orifice of the sac; or, not *directly* dependent on the aneurism itself, it may proceed from a spot of the vessel pressed upon and rendered narrow by the sac. On the other hand, the diastolic murmur seems due to the reflux of blood from the sac through its orifice; and as the force of the reflux current must be comparatively slight, so, as a rule, diastolic aneurismal murmurs are feeble: sometimes, where undue force is given to the back current by co-existent aortic regurgitation, the diastolic murmur, especially if the sac be very close to the origin of the aorta; acquires unusual intensity. When the entry of blood into the sac is murmurless, and its escape productive of murmur,—that is, when diastolic murmur alone can be heard, the peculiarity probably depends on some special condition of the orifice of the sac, whereby a smooth surface is presented to the entering blood, and a surface, roughened by moveable fibrine or otherwise, opposed to the receding current.

A number of conditions tend to intensify or enfeeble aneurismal murmurs. If the heart's action be very weak; if the sac be filled in great measure with fibrine; if it be incapable of much expansion; if the opening be very large (inasmuch as the current is then too free), or if the opening be at once very narrow and smooth (inasmuch as possibly the current is too small to generate notable sound),—under all these circumstances a sac, otherwise well constituted for the purpose, may fail to furnish murmur. On the other hand, great roughness of surface, neighbouring pressure, and the presence of good conducting material round the sac, will intensify, really or apparently, these murmurs. Mere sounds may sometimes be rendered murmurish by suspension of the breath for a moment,—a fact to which it is not easy to supply a satisfactory clue.

III. *Symptoms.*—(*a.*) The weight and flesh of aneurismal patients undergo very considerable reduction in prolonged cases; though exceptional instances, where the sufferer continues stout to the last, are occasionally met with. The centripetal or centrifugal progress of the aneurism does not always explain this difference; if, on the one hand, the greater suffering, attached to the former mode of progress, tends to produce rapid emaciation, the protracted, though less acute, misery endured in the latter, eventually works out the same result. It appears that extreme emaciation has sometimes been mechanically caused by pressure on the thoracic duct. There is not any attitude, posture, or mode of decumbency peculiar to the subjects of aneurisms of the arch, as a class and for a permanency; in bed the patient usually lies on the back, with the head moderately high. But for the relief of particular kinds of pressure, peculiar attitudes may be assumed; thus, where a sac presses on the trachea, the patient frequently raises or throws back the head suddenly, keeping it in this posture for a time so as to project the sac forwards from the windpipe. When paroxysmal attacks of dyspnœa, from bronchial or nervous irritation, occur, the sufferer sits up with his head supported on his hands, the elbows resting on the knees, or bends over the back of a chair, &c. The sleep is not affected by the aneurism itself; but if pressure exist, the ordinary slumbers are fitful, interrupted by starts, and frightful dreams. During the urgency of bronchitic and asthmatic seizures, the patient may pass night after night out of bed. The expression of the face varies: it may be calm and not indicative of suffering, except during paroxysms of dyspnœa, growing then, terrified and imploring; or habitually cross and irritable; or anxious and worn; or simply significant of profound distress. The differences in the original temper of patients modify their facial expression during this, as all other chronic diseases. The colour of the face may be to the last florid in the main, with slight lividity; or habitually livid; or in no single point remarkable; or pale, sallow, and cachectic-looking: the latter alone is in the least degree distinctive. (*b.*) The integuments generally are of no special tint; sallowness is, however, sometimes observed; sweating is not habitual, though I have known it a troublesome symptom, even where the lungs were sound. The lower extremities remain singularly free from œdema; a fact that may have its diagnostic application. Œdema of the base of the neck, the face, and the upper extremities, and one or both sides of the thorax, follows pressure of the superior cava, or one or both innominate veins. The peculiar spongy elastic fulness of the base of the neck, looking like a *collar* of flesh, due to capillary turgescence, is also observed. The integuments over the sac sometimes alone become œdematous, from irritation or mere distending pressure. (*c.*) The joints are not affected; no positive connexion exists between rheumatism or gout and aneurism. (*d.*) The lips full, tumid, and livid, the tongue œdematous at the edges, and of purplish tint, the mucous membrane of the pharynx, thick, livid, and coated with viscid secretion, when other signs of venous pressure exist, display, if these be absent, no peculiarity of appearance. Dysphagia, slight or severe, paroxysmal or permanent or both combined, or in some severe cases disappearing completely for a while, after having been a constant condition (a change sometimes explicable by relief of pressure through hæmorrhage from the sac), is a symptom of considerable frequency; more commonly attending the disease in the descending and transverse parts of the arch, it may be absent even in the former case (Brader, U. C. H.), and co-exist with aneurism affecting the right angle of the arch (Downie, U. C. H.). The intensity of dysphagia much depends on the general and local nervous susceptibility of the patient; a slight amount of pressure on the œsophagus will produce greater difficulty of deglutition in some persons, than actual destruction, by pressure, of the coats of the tube in another.* Blood may be discharged in large quantities, from rupture of the sac, into the œsophagus,—an event which, moreover, need not prove immediately fatal. Whether slight oozing of blood may take place by filtration into the œsophagus, and be discharged by sputation, without actual hæmorrhage, I do not know from experience. The appetite fails altogether, if the aneurism be the source of pain; commonly it is capricious. Blood in small quantities, darkened and otherwise altered by the gastric fluids, is, it is said, occasionally vomited, after having trickled into the stomach from filtration through the walls of the sac and œsophagus; blood of similar origin has, we are assured, been traced in the stools. The bowels are habitually constipated in advanced cases, probably from the patient's inability to take exercise; flatulence distresses many (even male) patients; piles and pruritus about the anus seem to be more frequent than in the average of persons of equal age. Ascites does not occur.

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* A perforation the size of a shilling may exist without the very least dysphagia occurring during life; though, too, the current of blood in the sac must have borne directly almost against the gullet. Clin. Lect., Brader, loc. cit., p. 119.

(*e.*) Painful, hoarse, clanging, laryngeal cough, laryngeal rhonchi, dry or moist, audible sometimes at a distance, and dyspnœa, at once habitual and increasing paroxysmally, coupled with various morbid states of voice, indicate deep disturbance, functional and organic, of the larynx. The speaking voice may be husky, muffled, cracked, and hoarse; or simply weakened, or tremulous and variable in note, or actually lowered in register. The hoarse variety appears to depend on chronic laryngitis, with diminished current of air,—itself, in turn, traceable to pressure on the trachea;—pressure on a main bronchus is not sufficient for the purpose. Œdema of the glottis, depending on congestion, venous or sub-inflammatory, has in some cases been found. Paralysis and atrophy of the muscles of one side of the larynx, coupled with flattening and compression of the recurrent nerve, explained extreme vocal feebleness in a case observed by Dr. Todd. Tremulousness and variation of note have been traced to simple pressure on, and displacement of, the trachea. Paroxysmal dysphonia is explained by irritative traction of the recurrent nerve. The trachea, chronically inflamed, where the subject of irritative compression, is tender to the touch; and stridulous breathing is in part due to these conditions. Various forms of pain are, or may be, felt in the chest. First, immediately over the aneurismal prominence, pain may be produced by mere distension, by local pleurisy, or by irritation of the intercostal nerves. Secondly, pain in the neck and arms, down to the finger-ends, is traceable to irritation of the branches of the cervical and brachial plexuses. Thirdly, pain of a peculiar gnawing, terebrating character, constant, but increasing paroxysmally from time to time, exists at the dorsal spine, if the vertebræ are undergoing absorption. Fourthly, local pain and tenderness over the sternum, is sometimes connected with periosteitis going on to suppuration. The second class of pains are of shooting, piercing, or stinging character, may be brought on by the slightest movement, even that of turning in bed, and are more or less paroxysmal. The gnawing pain in the back cannot, as some persons have supposed, depend on irritation of the roots of the spinal nerves, or it would, contrary to what is the fact, radiate in the course of their branches. The tenderness of an aneurismal prominence is sometimes extreme. Sometimes this is associated with a sensation of heat, perceptible also to the hand of the observer. In addition to all these sufferings, a feeling of fulness, weight, load, tightness, and oppression is experienced within the chest, coupled with a dread of movement, lest something should be displaced by the change of posture. There are patients who suffer seriously from spasmodic contractions of the diaphragm, or sensation of constriction round the base of the chest,—the obvious results of irritation of the phrenic nerves. The state of the respiration varies. If there be no pressure on the tubes, or irritation of these, or pressure or irritation of the vagi, recurrent, or pulmonary nerves, the breathing is calm. Where any of these conditions are present paroxysmally, the breathing is temporarily accelerated in proportion to their amount. Or, if they are permanent evils, the breathing is laboured, whistling, stridulous, audible at a distance, and the patient commonly points to the trachea as the source of difficulty. The number of respirations per minute almost always exceeds the average of health more or less,—I have found it range from twenty-four to fifty-six. The ratio of the pulse to the respiration is subject to great variation; thus, in one of the cases already referred to (Brader), the mean ratio throughout the time of observation being as 2·9 : 1, the extremes were as 5·3 : 1 and as 2·3 : 1. In this instance, the variations of broncho-laryngeal symptoms furnished a key to the rises and falls. The chest play is more or less confined. If a main bronchus be encroached on, or the mass of one lung diminished by pressure, the play on the corresponding side will be relatively deficient; and there may be special want of expiratory rather than of inspiratory power. Dyspnœa is an almost invariable symptom where the sac is of any bulk, though all sensation of the kind may positively be wanting where the sac is even huge (Harris's case). In many instances, the first symptom attracting the patient's attention, commonly increasing gradually in intensity, dyspnœa acquires, *cæteris paribus*, most intensity when the horizontal part of the arch is aneurismal. In addition to the pulmonary causes of the symptoms enumerated above, may be mentioned pressure on the auricles, pulmonary veins, or pulmonary artery, and passing accumulation of blood in the right cavities of the heart. Dyspnœa is prone, too, to occur paroxysmally at night, from accumulation of sputa, pressure on the trachea, produced by accidental movement into such positions as throw the sac against that tube, and probably from reflex action. By day, the act of deglutition sometimes induces a severe fit. Cough, rarely absent, may be loud, dry, and paroxysmal,—paroxysms of the kind sometimes terminate in a syncopal state, or, after great effort, are relieved by expectoration of a thin, watery fluid. Unless under the influence of accidental inflammation of the air-tubes, there may be no expectoration at all. Blood may be discharged through the trachea in different manners. The sac and windpipe undergoing an extensive rent, a tremendous flow of blood may take place, and kill instantaneously; or, syncope occurring after copious discharge, coagula form and plug up the opening for the time;—upwards of a quart of blood may be discharged under these circumstances without immediately fatal result: but temporary escape of the kind is a rare exception to the common issue of such ruptures. Or, lastly, the expectoration may be habitually tinged with blood, so as to produce the red-currant jelly appearance: such expectoration, which is very unusual, does not derive this character from blood filtrating from the sac, but from pressure on the vessels of the lung.* Moderate discharge of blood, by diminishing the size of the sac, and also by diminishing congestions, sometimes affords great temporary relief of symptoms. Certain physical signs connected with the lungs are worth attention. Where the trachea is pressed on, the supra-sternal region sinks in very deeply on inspiration; rhoncho-respiratory fremitus, in consequence of the powerful stridor of respiration, is carried to its maximum point. Pulmonary percussion-dulness comes of various influences exercised by the sac on the substance of the lung, displacement, condensation by pressure, and collapse from obliteration of bronchial tubes. Respiratory murmur may be deficient to almost suppression, from bronchial pressure, through part or the whole of one lung, while exaggerated respiration exists elsewhere. Condensed strata of lung yield bronchial respiration; and dry and moist bronchial rhonchi are audible. (*f.*) The *heart* is said to become hypertrophous, especially if the sac originate near the sigmoid valves; but such effect is by no means constant: I have known the heart quite within the limits of healthy size under the circumstances. In some cases, the relative sizes of the arterial outlets of the heart undergo perversion: in one remarkable instance (Downie), the pulmonary orifice, when opened out, measured two and a quarter inches, while the aortic reached four and three quarters. This state of things must seriously increase dyspnœa. Inequality of force and fulness of the radial, carotid or subclavian pulses occasionally exists. Though other explanations have been suggested, it appears that pressure or obstruction with coagula at the aortic origin of the weakly beating vessel is the only positive cause of the difference. The vessel on the affected, beats a little later, too, than on the sound, side. There is a certain sharpness and jerking character in the pulse, sometimes not unlike that of slight aortic regurgitation;—but the superficial arteries do not beat visibly. The pulse is sometimes bisferiens; however, observation does not justify the notion that the second wave depends on reaction of the aneurismal sac. The veins of the chest, of one or of both upper limbs may be enlarged, full, and knotty; sometimes so firm that they cannot be obliterated by pressure; those of the arm have been known to undergo complete obliteration by coagula. I have never seen pulsation of the jugulars in these cases. The situation of the obstructed veins will guide to that of pressure, whether on the superior cava or either innominate vein alone. (*g.*) The bronchial glands are sometimes enlarged, and increase the percussion-dulness of the aneurism in the back. (*h.*) The urine frequently contains excess of urea; it is free from albumen. (*i.*) The genital organs present nothing special. (*k.*) Cephalalgia is a frequent symptom, sometimes depending on the throbbing action of the arteries, sometimes simulated by pain in the nerves of the scalp, from pressure on the plexuses below. Paralysis of an arm has occurred from pressure on the brachial plexus. The intellect is unaffected to the last. (*l.*) Paraplegia has in rare instances followed erosive destruction of the vertebræ and pressure on the spinal cord.

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* Brader's case, loc. cit., p. 121.

IV. In the great majority of cases the progress of aneurism is slow and insidious at first: the symptoms may, however, be suddenly developed, probably from some sudden increase in the bulk of the sac,—an increase generally connected with change from the simple to the mixed condition of the disease. The further course of cases is either gradually or interruptedly progressive: occasionally aneurisms of considerable dimensions have remained latent to the last, and their physical signs been merely those of a soft, solid mass within the chest. Physically, the course may be centripetal or centrifugal, rarely both combined. The modes of death are by rupture of the sac (and there is no conceivable position into which the blood has not poured in different instances), by pure suffocation, by irritative pressure on the bronchi and trachea, by serous effusion into the pleura and pericardium, by exhaustion, by rupture of the aorta, &c.*

V. Tubercle and aneurism rarely co-exist in the same person; but the subject of the latter disease has nevertheless in some instances been cut off by phthisis. The difference of age, at which the two diseases are common, is probably the main reason of their rare association. The syphilitic cachexia and gouty diathesis are said to have some connection with aneurism: the fatty diathesis has a stronger claim to the character. A fanciful parallel has been drawn between aneurism and cancer. Like the cancerous, it is said, the "aneurismal diathesis" is never extinguished: what comes, then, of the cures, spontaneous and by art, of the disease? Frequently, we are assured, many of the arteries are involved in the same person: the assurance is directly at variance with statistical returns; besides, admitting the fact, the analogy fails; cancers multiply through the blood; aneurism could only do so through local changes in the vessels. The aspect of the patient and the general decay of the organism resemble those observed in cancer, according to this argument: the statement is decidedly inapplicable to the majority of cases of aneurism, and only true of those attended with an extra share of suffering. Besides, in their distribution to the two sexes, aneurism and cancer are well nigh the antipodes of each other.

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* Various rare causes of death may be found in Mr. Crisp's valuable collections of cases.

VI. The *diagnosis* of sacculated aneurism of the arch turns essentially on the co-existence of (1.) *a pulsating prominence*, visible and palpable, limited in area, and corresponding in seat to that portion of the vessel; (2.) the *signs of internal pressure;* and (3.) *certain arterial murmurs and sounds.* But a pulsating prominence may also be due to a small solid mass lying over and receiving the impulse of a sound aorta,—its pulsation may even be quasi-expansile; or to an abscess in the mediastinum; or to pulsating empyema; or to a tumour pulsating interstitially. Secondly, the signs of internal pressure may be produced by solid tumour. Thirdly, a double hoarse murmur over the arch, inaudible or only faintly audible at the heart,—a diastolic murmur similarly localised, and a pumping or sucking character of the aortic sounds, without murmur, are the most significant auscultatory signs.

Tuberculous consolidation of one apex, especially the left, with murmur in the subclavian or pulmonary artery, is distinguished from aneurism by the non-extension of percussion-dulness across the middle line; by its extension, on the contrary, to the acromial angle; by the existence of some tone in the percussion-sound, and some resilience in the wall of the chest; and by the absence of pressure signs, eccentric or concentric. The symptoms, local and general, are also different. *Fluid in the pericardium* is distinguished by the pyramidal form of its dulness, which aneurism never simulates except under the very rare accidental circumstances already referred to (p. 446). In a case of *enlarged heart*, there is but one centre of motion—in aneurism two; itself and the heart. If the sac be quiescent and non-expansile, however, the site of the percussion-dulness and the existence of pressure-signs must be appealed to for the distinction on the side of aneurism, and more or less of its special signs on the side of enlarged heart. If general dropsy exist, this is in favour of heart-disease, against aneurism. *Pulsating empyema*, with its throbbing prominence near the edge of the upper bone of the sternum, simulates aneurism closely, but may be distinguished by the rules elsewhere set down (p. 315).

A chronic sub-periosteal abscess of the sternum, forming a small prominence in the line of the arch, fell under my notice some time ago,* sufficiently resembling an aneurismal sac. But there was no impulse; gentle percussion immediately round the prominence gave clear resonance; there was no murmur, and concentric pressure-signs were totally absent. But there might accidentally have been impulsive action of the vessel beneath, and excess of mediastinal fat might have rendered the percussion-sound dull; under such circumstances the diagnosis would have been excessively difficult. *Infiltrated cancer of the lung* causes retraction of the side, produces no local prominence, deepens the intercostal spaces, and frequently renders the percussion-sound tubular in the infra-clavicular region; it does not produce pressure-signs, and may be the seat of the signs of softening and excavation. *Tumour* in the anterior mediastinum presents the greatest number of positive points of similarity to aneurism. Now, if there be highly-marked pulsation, a broad based prominence with conical elevation in the centre, the murmurs most distinctive of aneurism, and a sensation of the flow of liquid beneath the integuments, there can be no doubt that, whatever other grounds for diagnosing tumour may exist, aneurism is really present. But every one of these things may in cases of aneurism be absent: then, observe how like the two things are—a sac filled with fibrine and a solid tumour. In truth, one is a tumour *inside*, the other *outside*, the arch; and obstruction from without may have the same effect as from within on its circulation. Common to the two things are dulness and non-resilience (it may be, extending across the middle line), all the signs of concentric and all the signs of eccentric pressure. Under such circumstances, the question becomes one of pure probabilities. The conditions in favour of aneurism would be these: situation in the course of the arch, vibratile thrill above or below the clavicle, gradually increasing nearness of pulsation to the surface (but, from stratification of fibrine, the pulsation of an aneurism may grow deeper, and that of a tumour may become more superficial), absence of œdema of the arm and chest, dysphagia and great pain, especially of the dorsal spine. The circumstances in favour of tumour, and against aneurism, would be the facts of the patient being a female* and under twenty-five years of age; great superficial extent of percussion-dulness, especially if there were no marked attenuation of the walls of the chest; absence of any heaving motion in the affected spot; want of accordance between the sites of maximum-dulness and of pulsation; and currant-jelly expectoration (common with tumour, very rare with aneurism). It is a curious fact, that where a quiescent aneurismal sac and a tumour co-exist, the usually essential sign of aneurism, namely pulsation, apparently expansile, may be furnished by the tumour; and the usually essential sign of tumour, dead, pulseless dulness under percussion, may be caused by the aneurism. This statement is well illustrated by the case of Brader (loc. cit). The means of distinguishing *constriction of the aorta* have already been examined (p. 540).

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* Case of Marg. Mottlee, U. C. H., Females, vol. v., p. 36.

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* But this is of little value, for the excess of aneurism of the arch in males is by no means so great as that of aneurism of all arteries indiscriminately.

Group *b.*—*Fusiform dilating aneurisms.* The peculiarities of these aneurisms are, as compared with the sacculated, diffuseness of pulsation above and below the clavicle, visible and palpable, comparatively much less below than above, though even there, if anæmia exist, it may be very considerable; more thrill above, less below, those bones; rough, prolonged, rasping, whizzing or whirring murmur, systolic only, audible along the arch, and louder there than at the aortic valves, if they also be the seat of murmur. Concentric and eccentric pressure-signs are almost or completely wanting.

Anæmia and nervous excitement may simulate such dilatation; the results of percussion will distinguish the cases.

Group *c.*—*Mixed aneurism.* The sudden extension of dulness in the situation of a simple sac, coupled with similar increase of pressure-signs, especially if these conditions follow effort of any kind, make it probable that the inner and middle coats have given way, and the outer undergone additional pouching. But in the signs of a mixed aneurism, when actually developed, there is nothing special. The prognosis is rendered worse by the yielding of the inner coats.

Aneurism of the Descending Aorta.

The signs of an aneurism seated between the termination of the arch of the aorta and the diaphragm will, of course, vary somewhat with the precise portion of the vessel affected. If the sac be not of large dimensions, little is to be learned by inspection; however, in certain positions, in consequence of its lying behind the heart and pushing this organ directly forwards against the ribs or sidewards, the maximum cardiac impulse may be transferred from the apex to the base, and so-called diastolic impulse also produced. Posteriorly the hand may detect slight arching to the left of the spine; if the arched surface be the seat of the least impulsive action, the sign becomes one of importance. Dulness under percussion, limited to the same situation, and inexplicable by the condition of the lung, heart or pleura, would, of course, strengthen the inference drawn from the previous sources. An aneurism in this situation may supply the varieties of murmur and sound already enumerated; murmurs must be stronger over the sac than over the heart, to have any diagnostic value. Feebleness or deficiency of respiration in the spine, or over the side generally from pressure upon the main bronchus, will corroborate the other signs.

Vertebral gnawing and intercostal neuralgic pains, coupled with sensation of internal throbbing action (sometimes with difficulty distinguished from that of the heart) are observed in these cases. The laryngeal system, generally saved by its distance, sometimes suffers through extension of irritation from a main bronchus; the trachea, indeed, has, in rare instances, undergone direct pressure from very large-sized sacs. Perforation of the main bronchus, dysphagia from mechanical obstruction of the œsophagus, or, where the disease occupies the immediate vicinity of the cardia, various gastric symptoms simulating obstruction of that orifice of the stomach, have been noticed.

Death occurs more frequently, perhaps, from rupture into the œsophagus than from any other single cause; curiously enough, rupture into the right, occurs almost, if not quite, as frequently as into the left, pleura. The patient dies, worn out by the effects of bronchial and tracheal pressure in some cases. Aneurism of this division of the aorta is singularly rare in females.

The affection with which an aneurism, thus seated, may most readily be confounded, is hypertrophy of the heart; the strong systolic impulse, and the diastolic impulse combine to deceive. But careful employment of all the methods of physical diagnosis will prevent error in well-marked cases; while it must be confessed that very small sacs behind the heart, unless some accidental circumstances throw light on their existence, are exceedingly difficult of positive detection.

VII. In proceeding to the *treatment* of an aneurism of the intrathoracic aorta, the first point is to determine, if possible, whether we have a fusiform dilating aneurism, or one of the sacculated varieties, to deal with. For, whilst coagulation of the blood within the aneurism is scarcely obtainable in the former, and if obtained will not effect its cure; in the latter, there is a natural tendency to such coagulation, and coagulation does actually promote anatomical cure. Hence it appears that the clinical distinction of these kinds of aneurism is not a piece of mere scholastic refinement, as it has been slightingly called, but a matter of practical importance.

(*a.*) In cases of dilatation, the prevention of enlargement and rupture of the aneurism, may be best secured by occasional leeching over the affected part, especially if there be local tenderness, or by small bleedings from the arm. Full and repeated venesection, on the plan of Valsalva, is in this species of aneurism even less permissible than in the sacculated varieties; but a single abstraction of some ten or twelve ounces of blood is advisable at the commencement of treatment, more especially in plethoric persons. Purgatives, to such amount as to maintain a tolerably constant free action from the bowels, both from their sedative effect on the circulation, and from their preventing the necessity for effort in defecation, are essential. Diuretics do good service by keeping the quantity of fluid in the vessels below par. Direct sedatives of cardiac action, digitalis, aconite, hydrocyanic acid, and belladonna (internally and externally), lessen the violent pulsatile action of the diseased vessel, and may be given in various combinations, and more or less steadily.

The diet should be so arranged as to support, without exciting or over-nourishing: but anæmia is even more baneful than plethora, and the starvation system must be studiously avoided. The patient should abstain from all excitement, mental and emotional, pass the greater part of his time in perfect rest, and take but moderate daily exercise on foot: carriage exercise on smooth ground may be permitted to any amount desired.

(*b.*) In the instance of a sacculated aneurism, the object being to promote coagulation, venesection may be employed from time to time to lessen the force of the current. But there is a double danger to avoid here: if too much blood be drawn, the action of the circulating system will be excited, instead of tranquillised; and if the quality of the blood be seriously impoverished, the softness of the coagulum will probably render it comparatively useless as a support to the distended walls of the vessel. Theory, in truth, does not, at the present day, support the ideas of Valsalva concerning abundant depletion; and since the diagnosis of aneurism has become somewhat positive, cures obtained by his system have ceased to be heard of. Moderate leeching over the sac from time to time is always useful. Digitalis and other cardiac sedatives promote coagulation by enfeebling and slackening the current; and if there be no contra-indication in the state of the heart, a fair trial of them should never be omitted. Some practitioners have much confidence in the acetate of lead. Purgatives and diuretics are useful on the principles, a moment since, referred to; the latter especially because, while they diminish the water, they exercise no influence on the fibrine, of the blood.

Cold poultices of linseed meal and vinegar, or of conium and digitalis, relieve local suffering, and promote coagulation probably. Ice to the surface is grateful to some patients, unbearable by others: it can rarely be kept applied for a sufficient length of time to modify the circulation beneath. Cold poultices of oak bark have appeared to me, useful. If there come much pain over the sac, the application of the freezing mixture, as recommended for anæsthetic purposes generally by Dr. James Arnott, would, for a double reason, deserve a cautious trial.

Gallic acid, combined with digitalis, aconite, or belladonna, has appeared to me to exercise a very beneficial effect in promoting coagulation: it may be given in doses of two, three, and five grains twice or thrice daily, with occasional intermissions, for a length of time,—its constipating effects being obviated by occasional doses of castor oil.

The theoretical necessity for fibrine of good quality being clear in these cases, such diet is advisable as seems to promote its formation. However, the dangers of plethora, especially as much exercise cannot be permitted, must be held constantly in view, and over-nourishment avoided. Fluid in any quantity is injurious, and stimulants seriously baneful.

If the aneurismal sac be very superficial and ill-protected by fibrine near the parietes of the chest, it may be necessary to apply a shield, fitted to the part, to protect it from the chances of external violence.

ANEURISM OF THE ABDOMINAL AORTA.

I. Pulsating prominence, of variable extent, is seen anteriorly in the course of the aorta.* The hand, placed on this, receives a single systolic impulse, sometimes of enormous force and quite out of proportion with the volume of the moving mass, while posteriorly no trace of impulse may be perceptible. Generally speaking, the abdominal walls being thin, the hands may be passed on either side of the sac, and an estimate formed of its bulk: the impulse is felt to be laterally, as well as anteriorly, expansile. The chief pulsation may be to the right of the spine, the sac sometimes mainly growing in that direction. The mass is immovable, but compressible more or less: caution is, however, requisite in ascertaining these particulars. The left semi-circumference of the abdomen may, or may not, be increased.

The size of the sac can only be accurately estimated by percussion; and the tenderness of the surface and the neighbouring parts generally often interferes with the process.

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* Hallington, U. C. H. (vide p. 478.) Here, from one inch to the right of the umbilicus to the left anterior spine of the ileum.

I have heard in connection with aneurisms thus seated: 1. A single systolic murmur, without sound of any other kind; 2. A dull muffled systolic sound, convertible into a murmur by a little pressure; 3. A sharp, abrupt, short systolic murmur at the left lumbar spine, much more marked than in front; 4. A systolic murmur below the sac, none immediately over it; 5. Occasionally a dull second sound. I have never heard a diastolic murmur. In some instances systolic murmur is audible in the reclining, when inaudible in the erect, posture. Dr. Corrigan supposes this explicable by the removal of hydrostatic pressure in the former position, and the consequent greater freedom of the current in and out of the sac; but systolic murmur may be totally absent in every possible posture.

The size of the sac may be apparently increased by the extravasation of blood behind the peritoneum: this may occur on several successive occasions, and for some time before death (case of Hallington).

There may be a total absence of all positive physical signs, —neither impulse, murmur, nor percussion-dulness: the subjective symptoms are then very likely to deceive.

II. Pain following the course of nerves implicated by pressure —passing along the edge of the ileum down the thigh to the testicles, &c.—and in character raw, sore, pricking, cord-like, plunging, hot and burning or cold, accompanied with spasmodic difficulty in passing urine, and with tonic contraction of the flexor muscles and inability to straighten the limb, the whole accompanied with peculiar gnawing vertebral pain, existed in a case where the sac sprang from the bifurcation of the aorta, encroaching a little on the left common iliac. But obviously the neuralgic sufferings must vary with the exact site of the sac. Theoretically, anasarca of the lower limbs, or of one of them, must occur, according as the inferior cava or either iliac vein is pressed on; but in practice either effect is most rare. Wasting of the testicles I have seen from obliteration of the spermatic artery. Pressure on the descending or transverse colon may obstruct the bowels, and cause flatulence and great labour in defecation; yet there may be no hæmorrhoids (Harrington). The urine may be rendered albuminous by renal congestion induced by pressure on the emulgent vein; otherwise it is perfectly natural, as far as the aneurism is concerned. The respiration, if the sac is low down, is of natural frequency and character; when high, it interferes with phrenic action, throws the onus on the ribs, and accelerates the act somewhat.

III. Death may occur by rupture of the sac behind or into the peritonæum, into the pleura, lung, colon, renal pelvis, mediastinum, &c., or without rupture by jaundice, gangrene, exhaustion, &c. In some instances these sacs have acquired enormous bulk: one preserved in the Fort Pitt Museum is said to have contained ten pounds' weight of coagula. Hence the inference that the gradual growth of the disease is not incompatible with life.

IV. The difficulties sometimes arising in the distinction of mere *aortic pulsation* from aneurism have already been considered. *Fæcal accumulation* is distinguished generally by the oval outline of the fulness; by its doughy inelastic feel; by the existence of several spots of dull and clear resonance under percussion close to each other, and within the area of the swelling, from the intermixture of gas with solidified fæces; sometimes from the position of the mass; and, generally, from the history of the case. The pains of aneurism may be imperfectly imitated by those of peritoneal distension from the enlargement of the bowel; but it is rare indeed that a mass of fæces receives such arterial impulse from behind as to simulate that of aneurism. In the obscurity of their early symptoms, in the eventual pain, and in the gradual exhaustion they produce, there is considerable similarity between *lumbar and psoas abscesses* and aneurism; but the swelling of these abscesses passes in an elongated form from above downwards, and does not exhibit an irregularly globular one like aneurism: they give neither impulse nor murmur. Tenderness exists in the lumbar spine, and there may be loss of motor power in the lower extremities; but the actual pain is materially less, as a rule, than in the aortic disease. Tubercles should be sought for in the lungs: their presence would be directly in favour of lumbar abscess (of tubercular origin); against aneurism. *Hydronephrosis and pyelitic distension* are accompanied with renal symptoms, changes in the urine, tumour with the character of renal enlargement,—a tumour of tuberous nodular outline, non-impulsive, murmurless, and extending further into the flank and into the back than aneurism. The urine may be albuminous in all three affections.

V. The *treatment* is the same as of intra-thoracic aneurisms in general. Were the disease diagnosticated at an early period, might any good be effected by pressure either on, above or below the sac?—a cautious trial of one or other form of pressure might with propriety be made.

Group *d. Dissecting Aneurisms.*—The morbid anatomy of dissecting aneurism of the aorta, in its three essential varieties, is clearly demonstrable from existing records: its clinical history has yet to be worked out. And, indeed, from the nature of things it seems singularly unlikely that any general account, applicable even to the majority of such cases, can be given,—seeing that the symptoms must in great part depend upon the extent and precise portion of the aorta affected.

The symptoms in recorded cases may clearly be referred to three heads, which the observer should always aim at severally distinguishing;—namely, (1.) symptoms of shock to the system at large; (2.) of dynamic disturbance of the artery; and (3.) of mechanical interference with function. (1.) The symptoms of shock are, primarily, sudden faintness or actual syncope, and, on recovery of consciousness, nausea, vomiting, and pain in the thorax or abdomen: secondarily, febrile action (by no means necessarily very marked), thirst, furred tongue, abdominal tympanitis. (2.) The dynamic disturbances of the artery are signified by more or less severe pain in its course, and throbbing action, irregular in force and rhythm. (3.) The symptoms of mechanical origin are produced by the accumulation of the blood, filtrated between the coats of the aorta, against the orifices of arterial branches, whereby these are completely, or almost completely, blocked up. The nature of these symptoms will, of course, depend on the distribution of the blocked-up vessels. Thus, in a remarkable case, observed by Dr. Todd (Med. Chir. Trans., vol. xxvii.), where the innominate and the renal arteries were mainly obstructed, very singular cerebral symptoms and suppression of urine marked the event.

The obstruction to the current, offered by the prominent and ragged lining membrane in the site of its ruptures, gives rise to blowing systolic murmur, which, if seated near the heart, may be mistaken for that of constrictive disease of the aortic orifice. If a main bronchus were pressed on, there would be sudden deficiency of breathing, with clear percussion-sound.

Were the practitioner fortunate enough (guided by the sudden supervention of symptoms of the three classes now distinguished, and of a strong arterial murmur in a person known to have previously been free from this physical sign,) to divine the occurrence of acute separation of the coats of the aorta, it does not appear that, in the present state of knowledge, the treatment would be materially improved by his sagacity. Did he fail to diagnose the occurrence, his aim would be to recover the patient from the first shock of the accident, control excited arterial action, and relieve symptoms as they arose. And it does not appear that art could do more than this, were the anatomical nature of the affection understood from the first.

APPENDIX.

Vide pp. 15 and 17.

The observation of a considerable number of female children, between the ages of four and ten, who had never worn stays, leads me to the conclusion that, independently of any extraneous influence, there is proportionally more infra-clavicular movement in the female than in the male. But indubitably the main source of the excess of infra-clavicular movement in the adult female is due to the use of apparatuses interfering with inferior costal and phrenic action. The male dog breathes exactly like the human male, almost wholly with the abdomen, the chest rising inferiorly after the abdomen; the action in the female dog is very closely the same.

Vide p. 21.

Although convergence of the upper ribs during inspiration cannot be established clinically, I do not mean to contest its reality. On the contrary, I have found, by performing artificial respiration after the removal of the integuments, on the dead subject, that the upper ribs do actually converge during that movement. The amount of approximation, even in persons with wide intercostal spaces, appears to me not to exceed one-sixteenth or one-twelfth of an inch at the outer edge of the costal cartilages.

Vide p. 27.

Mr. Henry Thompson, of University College, has recently suggested a very simple addition to the tape-measure described in the text, whereby the absolute and relative expansions of the two sides of the chest may be ascertained during *one* and the same respiration. The more ordinary plan requires two, and as no two respirations are probably precisely equal, Mr. Thompson's instrument (which may be had of Coxeter, Grafton Street) obviates a source of fallacy.

Vide p. 289.

Since the description of pleurisy was printed, I have observed two points in its physical diagnosis to me altogether novel: perfect *cracked-metal note*, in the first and second interspaces, during the height of effusion and subsequently (case of J. Denley, U. C. H., Females, vol. vi., p. 85); and loud rubbing redux friction sound, with four or five jerks in inspiration and expiration, *above the clavicle* (case of G. Whiles, U. C. H., Males, vol. vi., p. 122).

Vide p. 405.

John Stoner, ætat. 25, admitted U. C. H. for second time, third stage of phthisis, April 10, 1849. The chest-disease gradually advancing.—*June* 2. Loss of power of speaking, semi-stupor, with difficulty roused, dysphagia, drink pouring back through mouth (not nose), pupils sluggish, no distinct convulsion, lower extremities drawn up, no paralysis, sensibility blunted, fixed frontal cephalalgia; seems conscious of all that is passing around him; P. 102, R. 44.—*June* 4. Since this seizure cough almost gone, scarcely any expectoration; alternate pallor and flushing of cheeks; no rigidity or convulsions, no screams, no strabismus; complete insensibility of dorsum of hands; tendency to rigidity of knee-joints; fingers firmly clenched, when unclenched by bystander contract again; P. 142, R. 44.—*June* 8. Sensibility returned, still speechless.—*June* 9. Spoke as usual for first time. This man was treated with mercurials, but not to ptyalism. Discharged on July 13th, he was readmitted August 18th, and died of his pectoral disease, the cerebral functions being perfectly natural, on October 11th, 1849. *Post mortem:* No morbid appearance at fissures of Sylvius; over both anterior lobes fine florid injection; numerous granules, opaque, greyish-white, size from pin's point to pin's head, lie under cerebral arachnoid of these lobes; convolutions here appear drawn together, as if from deep-seated contraction in the sulci; membranes here generally opalescent; dura-matral arachnoid adherent posteriorly to cerebral arachnoid of left hemisphere; membranes thicker than natural; anterior convolutions at the curve forwards, especially at left side, somewhat opaque; those of left side not materially softened,—those of right firmer than natural.

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